THE DENTAL PRACTITIONER

AND DENTAL RECORD

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VOL. IX, Nos. 11, 12 July–August, 1959

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THE DENTAL PRACTITIONER
AND DENTAL RECORD

Vol. IX, *Nos.* 11, 12 — *July–August*, 1959

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Vol. IX, Nos. 11, 12 July–August, 1959

GUEST EDITORIAL

YOUTH AND AGE

It is probably true to say that nearly all our patients require some form of advice on their dental condition, quite apart from the question of treatment. It is unfortunate that so little surgery time may be spent in the giving of advice to them. The economic position of dentistry tends to preclude advising patients, as no payment may be made for this type of service, and in any case there are few practices that are not overburdened with routine treatment.

The two large groups of patients who really require advice about the health of their teeth and mouth are the young and the aged—advice on the foundation teeth of a child and advice to those who have unfortunately lost their teeth and have to wear dentures. The small child who appears at the surgery with rampant caries and the patient with dentures he cannot wear are problems that sometimes require more guidance and advice than actual treatment.

The problems of dental health and dental treatment are too large for the general practitioner even to begin to advise on an individual basis. Advice by word of mouth may take up to half an hour over two, or even three, appointments. It can only be started by the printed word being presented to each patient. There are, of course, many pamphlets issued by the General Dental Council, the Ministry of Health, and the Oral Hygiene Service, as well as by other bodies, which are of great help.

We would like to welcome two new editions to the range of small books which, we feel, should be in every waiting-room but could be recommended to the patient for reading—*Your Child's Teeth*, by Edgar Bacon (edited by Professor Stones), and *New Teeth for Old*, by Victor Sears. Both these books are written for and designed to help the patient. Their recommendation to the patient will ensure that the essential advice for the young child and the denture wearer will be conveyed in a simple, readable form. Both books take a commonsense view and will undoubtedly help the patients to overcome many of their dental faults.

Perhaps one day in the future the Dental Profession will find itself in a position whereby it can give advice in the same way as the medical practitioner advises his patients. However, until that day arrives, we must present our advice in a different form, providing we realize that one of the essential parts of a dental service is to advise the patient as well as to treat him. N. L. W.

THE EFFECTS OF ABRASIVE AGENTS ON AN AMALGAM SURFACE

By **J. R. GRUNDY**, B.D.S. (B'ham)
*The London Hospital Dental School**

BOTH Anderson (1956) and Skinner (1956) in their books on dental materials cover a wide range of abrasive and polishing agents, and they give detailed information on the theory of abrasion and polishing. However, no specific recommendations are made regarding the polishing of amalgam. Similarly, textbooks on metallurgy treat the subject in a general way only.

Although it is generally agreed that 3 or 4 abrasives with different qualities are required to produce a high polish on amalgam, considerable variation of opinion exists as to what are the best agents to use and in what order they should be applied.

It is generally accepted that production of a progressively smoother finish on the surface of the amalgam restoration is attained by application of abrasives in descending order of abrasiveness. To determine this order, a method was devised to assess the abrasive qualities of certain agents commonly in use.

The scope of this study is being extended to include other abrasives; to observe the effects on the enamel adjacent to the filling; and to determine the effect of variations in the quality of the amalgam itself.

METHOD

It was found unsatisfactory to apply the various agents one after another to the untreated surface of an amalgam specimen, as would be done in clinical practice. First, the surface soon became uneven so that sharp focusing at high magnifications was impossible. Secondly, succeeding agents would not be tested under identical conditions; the state of the surface upon which each had to work being dependent on the abrasive qualities of the previous agent.

In order to provide each agent with exactly similar conditions of test, a highly polished plane surface was prepared on the amalgam specimen before application of the abrasive.

It was reasoned that for any particular agent the same quality of surface would be produced by roughening a smooth surface as by smoothing a rough one. In both cases, the result would be the best that could be expected from the abrasive under consideration.

The quality of surface produced by each abrasive was assessed by microscopic examination, and the surfaces varied so distinctly in appearance that it was possible to place them in order of smoothness without any difficulty.

The Amalgam Specimen.—The same amalgam specimen was used throughout the series. It consisted of a cylinder 4×8 mm. made from an alloy conforming to the A.D.A. Specification No. 1. The amalgam was condensed into a cylindrical steel mould, first with hand pluggers, and then followed by a mechanical condenser of the vibrator type. The hand pluggers were 1 mm. and 2 mm. in diameter, used with a thrust of approximately 10 lb. The diameter of the vibrator point was 3 mm., and this was applied with an engine speed of 5000 r.p.m., resulting in 20,000 vibrations per minute.

The specimen was, therefore, as well condensed as any amalgam restoration obtained under average clinical conditions.

One week after the specimen was made it was mounted in a cold-curing mounting plastic. As it was likely that variations in hardness existed throughout the length of the specimen it was mounted so as to present a longitudinal section to the abrasives. In this way each abrasive was applied to a comparable surface.

The Initial Polish.—The mounted specimen was taken down with silicon carbide papers, well lubricated with white spirit, until approximately one-third of the cylinder had been removed. A high polish was then obtained on

* Present address: Turner Dental School, Manchester.

a polishing machine, using Selvyt cloths impregnated with proprietary metal polishes, first "Bluebell" and finally "Silvo". The resulting surface is depicted in *Fig.* 1.

Method of Abrasion.—Each abrasive was applied to the amalgam for 5–10 seconds, using a contra-angle handpiece on a dental engine working at 5000 r.p.m. The surface

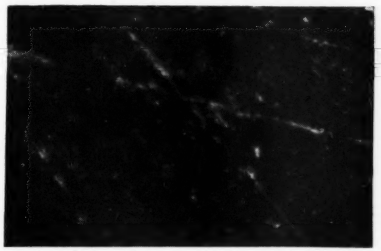

Fig. 1.—The amalgam specimen after mechanical polishing with "Silvo". (× 218.)

Fig. 2.—A highly polished gold surface showing a few light scratches. (× 218.)

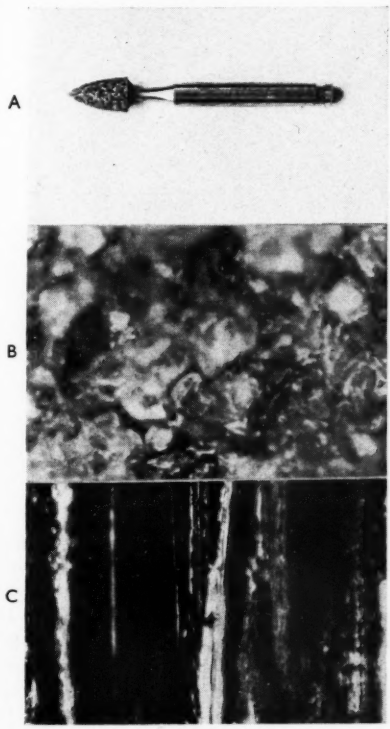

Fig. 3.—A, Carborundum stone. B, The surface of the carborundum stone. (× 30.) C, Amalgam surface after application of carborundum stone. (× 218.)

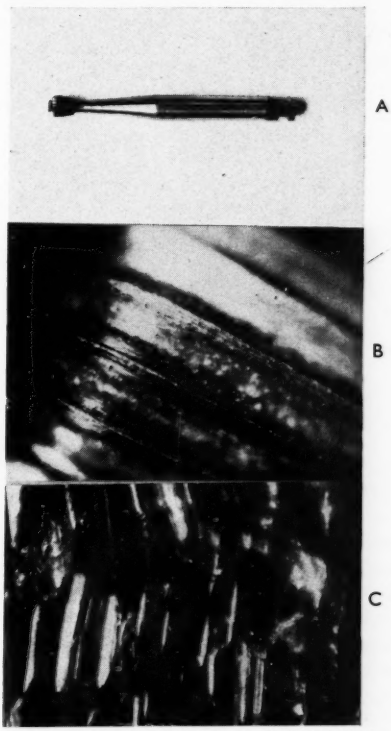

Fig. 4.—A, New finishing bur. B, New finishing bur. (× 30.) C, Amalgam surface after application of the finishing bur. (× 218.)

was re-flattened and re-polished before each application.

The abrasives used were carborundum stone, new finishing bur, worn finishing bur (*see Fig.* 8 B), pumice and petroleum jelly (applied both with a bristle brush and with a rubber prophylactic cup), and whitening powder and alcohol (applied with a bristle brush and with a rubber cup). Each was applied once in this order and the series was then repeated twice more.

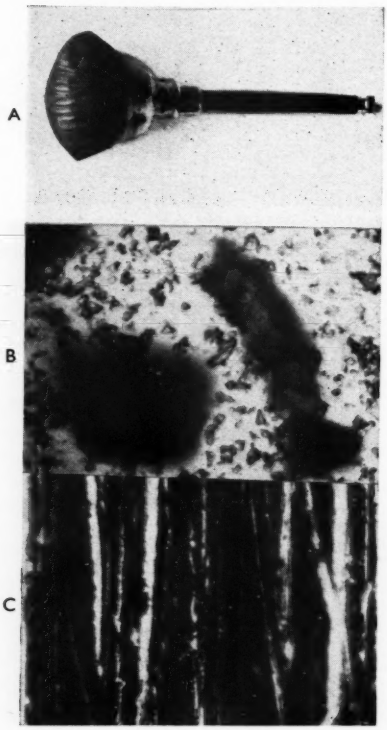

Fig. 5.—A, Bristle brush. B, Pumice powder. (× 96.) C, Amalgam surface after application of pumice with bristle brush. (× 218.)

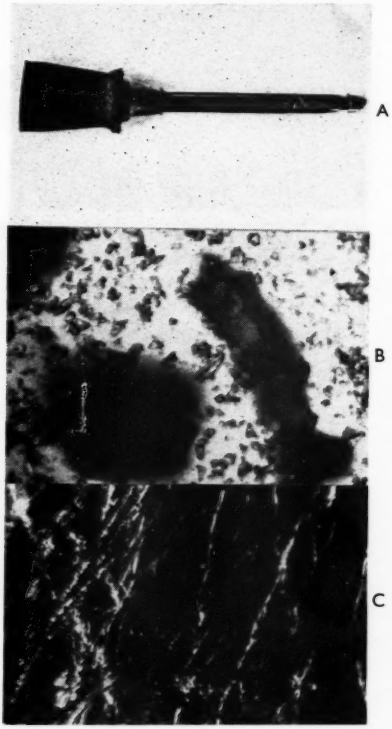

Fig. 6.—A, Rubber cup. B, Pumice powder. (× 96.) C, Amalgam surface after application of pumice with rubber cup. (× 218.)

MEANS OF OBSERVATION

The Leitz "Ultrapak" system was used. This provided incident illumination, the light of which struck the object obliquely. The angle of incidence was so arranged that for a truly flat surface all the light was reflected outside the aperture of the objective lens and the object then appeared to be completely black when viewed through the eyepiece. *Fig.* 2 demonstrates this by showing a gold surface with a near perfect polish. A few light scratches show as very faint lines, the highly polished part remaining black. When the surface being examined is not completely flat it presents an irregular surface to the incident light, which results in a certain scatter of the reflected light. Some of the scattered rays will enter the objective lens for observation and these can be photographed.

RESULTS

Each abrasive produced a typical surface on the polished amalgam specimen, which was virtually the same on each of the three occasions that the abrasive was used. *Figs.* 3–9 show the abrasives and their effects. The first photograph in each figure shows the natural size of the abrasive agent or its means of application. The second shows a photomicrograph of the agent and the third a photomicrograph of the amalgam surface after the application of the agent.

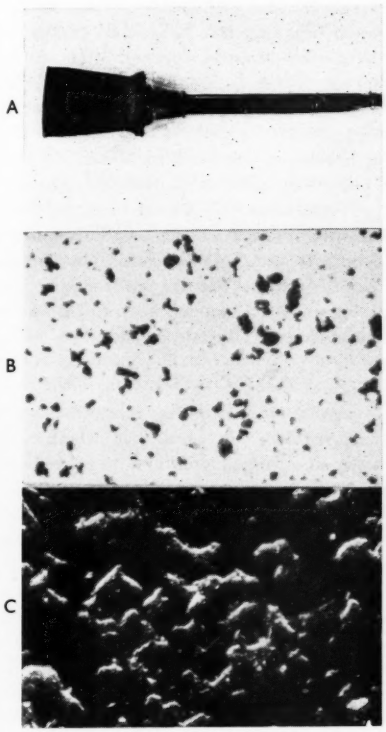

Fig. 7.—A, Rubber cup. B, Whitening. (× 96.) C, Amalgam surface after application of whitening with rubber cup. (× 218.)

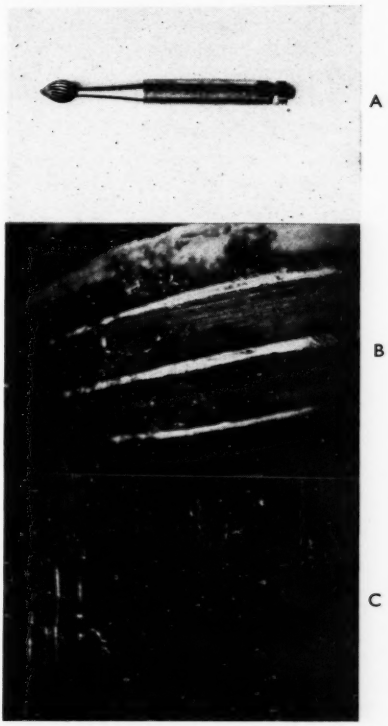

Fig. 8.—A, Worn finishing bur. B, Worn finishing bur. (× 30.) C, Amalgam surface after application of the finishing bur. (× 218.)

Carborundum stone is seen to be very coarse in texture (*Fig.* 3 B), and this is emphasized by the impossibility of getting all parts of the surface sharply in focus at the same time. As would be expected the resulting amalgam surface (*Fig.* 3 C) is scored with deep grooves, and sharp focusing is not possible due to the level differences between the depths of the grooves and the crests between them.

The surface left by a new finishing bur (*Fig.* 4 C) is little better than that produced by carborundum. It has a distinctive pattern composed of clusters of short parallel scratches, and a point of interest is that the clusters do not have one common axis, but are set at varying slight angles to one another. This is thought to be due to the "chatter" experienced when the new finishing bur was applied to the amalgam. This occurred despite the fact that

there was no play in the handpiece, and was in marked contrast to the smoother running of the carborundum stone and the worn finishing bur.

The pumice powder used in this series of tests has an extensive range of particle size

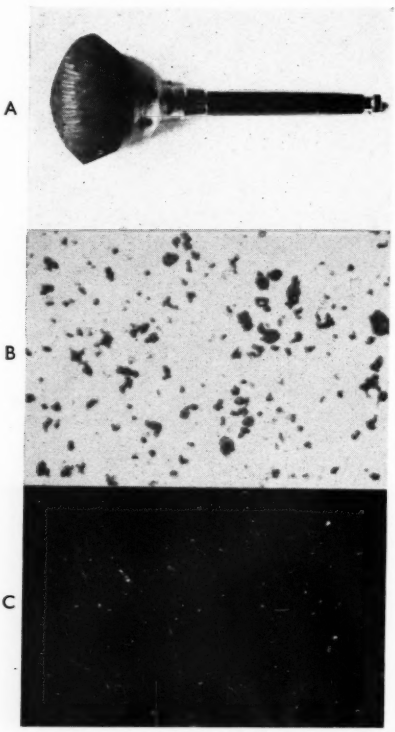

Fig. 9.—A, Bristle brush. B, Whitening. (× 96.) C, Amalgam surface after application of whitening with bristle brush. (× 218.)

(*Figs.* 5 B, 6 B). When used with a bristle brush (*Fig.* 5 A) it produced an amalgam surface composed of continuous scratches (*Fig.* 5 C) which are both shallower and narrower than those made by carborundum. When pumice was applied with a rubber cup (*Fig.* 6 A) a characteristic criss-cross pattern appeared which is shown in *Fig.* 6 C.

The particle size of the whitening is small and very even (*Figs.* 7 B, 9 B), though there is a tendency to clumping, which gives the false impression of larger particles being present. When whitening is applied with a rubber cup, the amalgam surface is raised into distinctive wave-like mounds (*Fig.* 7 C). With a bristle brush, however, a very high polish is obtained (*Fig.* 9 C) comparable to that produced by the polishing machine (*Fig.* 1), with only a few light scratches still evident.

A worn finishing bur (*Fig.* 8 B) produces a smooth surface, faintly marked with shallow parallel scratches. It should be noted that this surface is smoother than that produced by the pumice.

The abrasives depicted in *Figs.* 3–9 have been placed in order of descending abrasiveness, as determined by visual examination of the resulting amalgam surfaces. This order is: carborundum stone, new finishing bur, pumice and bristle brush, pumice and rubber cup, whitening and rubber cup, worn finishing bur, and whitening and bristle brush.

CONCLUSIONS

The agents subjected to test are seen to produce surfaces on amalgam which differ markedly in a range from very rough to highly polished.

Whitening and alcohol, when applied with a bristle brush, results in a standard of polish which is quite high enough for clinical purposes. However, it is known from clinical experience that whitening does not possess enough abrasive power by itself to take an unpolished amalgam through to the final polished state. Some other agent is required to level the rough amalgam restoration sufficiently to enable the whitening to polish effectively. A worn finishing bur is found in practice to do this very satisfactorily. It is capable of working efficiently on "raw" amalgam, and *Fig.* 8 C shows that the resulting surface is smooth, with only a few light scratch marks to be removed by the whitening.

It is, therefore, concluded that a worn finishing bur, followed by whitening applied with a bristle brush, will produce a polish on an amalgam restoration, and this has been found to be so in clinical practice.

Carborundum stone produces such a rough surface that its use is contra-indicated as a

prelude to polishing. However, it is known to remove amalgam speedily and is therefore sometimes needed when gross modifications to the contour of an amalgam restoration are required.

A new finishing bur also removes amalgam efficiently, but it will leave a very rough surface. A smoother surface is obtained if the bur is blunted before use.

Pumice of the grade used in this series of tests makes a rougher surface than does a worn finishing bur and it would appear to be unnecessary for normal routine polishing. However, if a carborundum stone or new finishing bur has been used, pumice may be desirable as an intermediate abrasive before the final polish is attempted. It is well known that abrasives should not be applied with too great a difference in abrasive qualities between one abrasive and the next.

The irregular wave-like mounds worked up on the amalgam by rubber cups would seem to contra-indicate the use of rubber cups as a means of applying abrasive pastes.

RECOMMENDATIONS

By relating these findings to clinical practice the following recommendations for speedy and efficient polishing are made:—

1. Careful carving at the time of filling, which will make the later use of carborundum stones and new finishing burs unnecessary.

2. Removal of marginal edges, slight adjustments to contour, and general smoothing with a finishing bur that has been well blunted.

3. Polishing with whitening and alcohol applied with a bristle brush.

This method has been used routinely by the author for some considerable time and has produced highly polished amalgams rapidly and easily.

SUMMARY

A standard specimen of amalgam has been subjected to a number of abrasive tests under standard conditions. The results of these tests have made it possible to place the abrasives in an order of abrasiveness. Recommendations are made for polishing an amalgam restoration efficiently and speedily.

Acknowledgement.—This work was given financial support by the Yarrow Research Fund of the London Hospital, and this is gratefully acknowledged.

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ANDERSON, J. N. (1956), *Applied Dental Materials*, 1st ed. Oxford: Blackwell Scientific Publications Ltd.
SKINNER, E. W. (1956), *The Science of Dental Materials*, 4th ed. Philadelphia: W. B. Saunders.

Alveolar Bone Loss as related to Oral Hygiene and Age

A radiographic assessment of marginal bone loss in the alveolar process was made in 737 male members of staff of a modern industrial plant in Norway. Measurements were made by means of a transparent ruler on which radial lines had been drawn to enable an immediate calculation of the percentage of bone loss. The base line was taken as the cemento-enamel junction, and the normal bone margin to be 1 mm. apical to this. Where any difficulty was encountered in finding the cemento-enamel junction the site was not measured.

Oral hygiene was graded as good when all, or practically all, surfaces accessible to the toothbrush were clean; fairly good when some of the surfaces accessible to the toothbrush were clean; and not good when practically all the surfaces accessible to the toothbrush were covered with deposits and could have been removed by brushing. It was found that:—

1. Bone loss increases with decreasing efficiency of oral hygiene. The difference was not very marked in the 21–25 year age-group, but became more apparent in the later age-groups.

2. Bone loss increases with age.

3. The pattern of bone loss showed that it was greater around anterior teeth and decreased around molars, premolars and incisors.

4. A higher incidence was found around the maxillary right incisor teeth, it being suggested that this was due to neglect of this area by those using their right hand in brushing.—SCHEI, O., WAERHAUG, J., LOVDAL, A., and ARNO, A. (1959), *J. Periodont.*, **30**, 7.

THE BIFID SPOON DENTURE

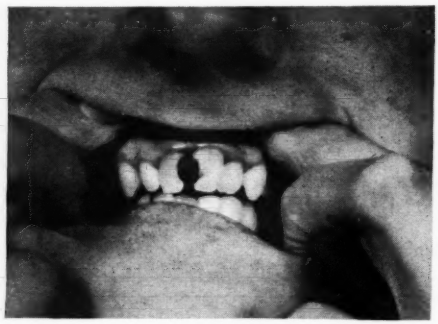
A SURVEY OF THIRTY-FOUR CASES

By **G. M. RITCHIE**, L.D.S. R.C.S.

Assistant to the Professorial Unit and Lecturer in Prosthetic Dentistry, University College Hospital Medical School

THE type of denture to be described is intended for the replacement of one or more upper anterior teeth.

From the saddle area the denture base extends distally along the hard palate, dividing in the region of the rugæ into two long "fingers" which abut on to the lateral walls of the palate. Both the palatal gingivæ and the vault of the palate are left exposed. Labial gum work is usually fitted to obtain better retention.



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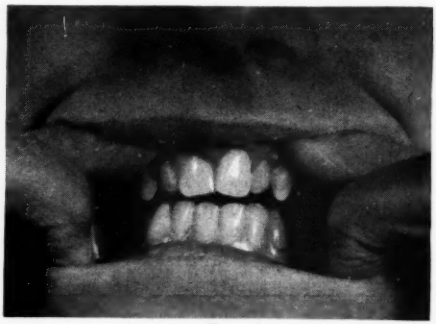

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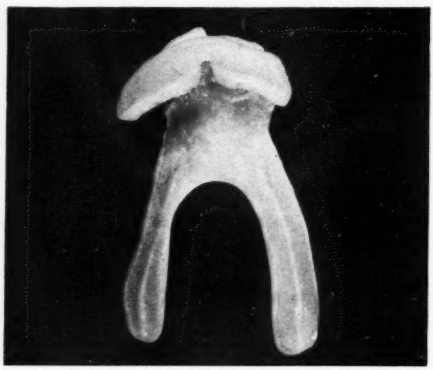


C

Fig. 1.—A, Girl, aged 13 years, with carious $\underline{1|1}$ and gross periapical infection: B, Bifid spoon denture immediate insertion of $\underline{1|1}$. Labial gum carried to maximum extent. C, Bifid spoon denture replacing $\underline{1|1}$. Note bilge keels and pin dam.

SELECTION OF CASES

Cases where one or more upper anterior teeth are to be replaced are suitable for this method of treatment, except those presenting with a very flat palate. Narrow, high-vaulted palates are particularly favourable for retention. A large torus palatinus provides no difficulties, since the bifurcation of the denture palate will skirt this area.

Immediate insertion is similarly no contra-indication, but the prognosis for plates carrying more than three teeth is unlikely to be favourable (*Fig.* 1).

THE MODE OF RETENTION AND STABILITY

Six factors play a part in the successful retention and stability of the dentures.

1. Frictional contact between the denture base with replacement teeth and the interstitial surfaces of the abutment teeth.

2. By using a compression–impression technique the fatty tissue underlying the mucous membrane on the lateral walls of the palate may be displaced or compressed. The elastic recoil of the soft tissues on the fingers of the

denture when in position in the mouth will have the effect of springing the denture into position.

3. "Bilge keels" are made on the fitting surface of the fingers, extending from the region of the rugæ to the distal end of the denture. These are longitudinal ridges corresponding to the most compressible tissue on the lateral walls of the palate.

The keels subserve the same function as those fitted to boats, namely preventing lateral and anteroposterior rotation or rock. In addition, they prevent downward displacement of the distal ends of the denture when a biting force is applied to the replacing teeth. A third function may also be ascribed, which is preventing the soft tissue recoil from the displaced tissue pushing the denture up the inclined plane formed by the sloping sides of the palate.

4. Labial gum work is advisable to obtain additional surface area and to prevent backward displacement of the denture. The undercut area often present near the base of the ridge may be utilized to aid retention by preventing displacement vertically downwards.

5. The peripheries of the palatal portion of the denture should be pin-dammed to provide a better peripheral seal and to allow the edges of the "fingers" to be thinned to a minimum.

6. Rests should be provided on the abutment teeth to prevent sinking and anteroposterior rock. They will also assist in guiding the denture into the correct position in the mouth.

CLINICAL TECHNIQUE

An impression is taken in a stock tray with an elastic composition material and removed from the mouth whilst still soft.

The periphery must extend to full functional depth of the sulcus in the region of the edentulous saddle. The peripheries of the impression should then be expanded a little and the composition chilled. Teeth and gingivæ are cut out, using a flame vulcanite bur or wax knife, and the impression replaced in the mouth with a little wax or greenstick tracing composition added in the regions of the intended fingers of the denture. The impression is again removed, chilled, and dried. The lateral walls of the palate are palpated to find the area of maximum compression and the position of the bilge keels marked in the mouth with an indelible pencil.

The impression is now replaced with an alginate wash, and from the final result the working model is obtained. An impression of the occlusal surfaces of the lower teeth is obtained and the bite registered with a wax leaf. Tooth and gum shade are also recorded.

LABORATORY TECHNIQUE

The cast produced from the upper impression will bear the indelible marks denoting the area of greater compressibility. These will show the position at which the bilge keels will be cut into the cast. An outline of the future denture is inscribed on the model in pencil. The denture extends backwards from the saddle, dividing into two fingers in the distal region of the rugæ, terminating at the line joining the distal surfaces of the second molars.

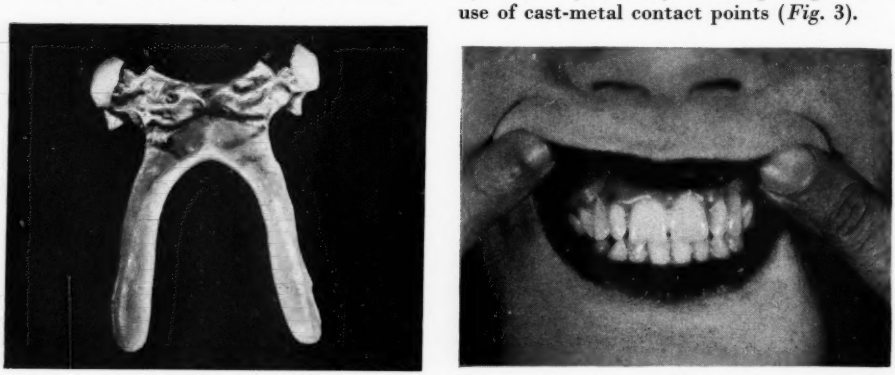
The gingival peripheries of the fingers should approach no closer than 4 mm. from the palatal gingivæ of the maxillary teeth. Thickness of the fingers should be about 1 mm. and the width about 15 mm. Peripheries are pin-dammed and then bilge keels cut approximately 1 mm. deep and at an angulation of about 40° to the gingival half of the fitting surface. They should extend from the distal termination of the rugæ to join the post-dam line at the distal ends of the fingers. The model is surveyed to allow the maximum undercut, especially in the labial sulcus, to be utilized for gum work, and whenever possible this should extend laterally to lines drawn through the median longitudinal axis of the abutment teeth.

Occlusal rests should be drawn in with reference to the bite, and in cases of close or deep overbites casting in gold or cobalt chrome may be necessary (*Fig.* 2).

The denture should be waxed up as for a conventional spoon denture, using one layer of wax. This enables the technician to deflask

the denture with greater ease and with less chance of fracturing one or both of the fingers. After trimming off the flash, the centre portion is cut out prior to polishing.

No try-in of the waxed-up denture is necessary, but when packing the case for finishing, a trial closure procedure should be adopted. At this time radiopaque material should be included.



A B

Fig. 2.—A, Bifid spoon denture to replace $\underline{3|3}$ incorporating cobalt chrome bifid saddle. B, Denture in position in the mouth.

RADIOPACITY

These dentures, although not large, are of sufficient length and width to make the possibility of them being swallowed most unlikely; but due to the thinness of the fingers it is possible that a fracture of the hairline type might occur if the denture is dropped during cleaning. A complete fracture might later occur in the mouth, with the chance of a fragment being swallowed or impacted in the throat. Radiopaque material should therefore be included in the form of small disks of gauge 4 relief metal (tin), or stainless steel rings, which may be obtained by cutting up a denture spring.

ÆSTHETICS

Success will largely depend on how closely it has been possible to copy the conditions existing in the mouth before the teeth were removed. The replacement teeth should be a replica of the natural predecessors, or of the standing partner in size, form, shade, and inclination. Character marks should be included, and facets ground to accommodate movements of the mandible. These will assure a more natural appearance. Diastemas and large interdental gaps may be simulated by clear acrylic, or by actual spacing and the use of cast-metal contact points (*Fig.* 3).

Extension of the gum-work to the greatest possible width has been mentioned for its importance previously, and the appearance may be improved by contouring and stippling, with inclusions of veined acrylic of the nylon thread type. A very suitable shade of acrylic for coloured patients may be obtained by adding a varying amount of gold dust to pink acrylic. This imparts both a flecked appearance and a blue tinge to the pink. Care must be taken to polish only lightly to prevent the dust standing proud of the acrylic surface.

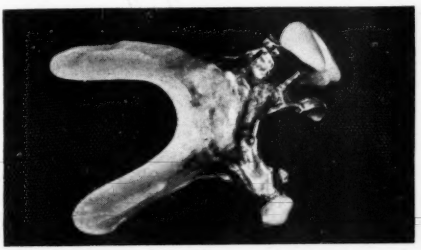
ADVANTAGES

A small denture gives freedom to the tongue, and hence does not interfere with speech. Leaving much of the palate uncovered allows appreciation of a greater delicacy of flavour in the diet.

The æsthetic appearance and retention are good, allowing most patients to bite on the replacement teeth without dislodgement. Gingival irritation and food trapping do not occur since the denture is kept well away from the palatal gingivæ. No specialized conservative

techniques or occlusal grinding are required to accommodate cast rests and clasps.

Conventional spoon dentures tend to be unsatisfactory on palates where a large torus palatinus is present. Even those that are well retained when first inserted soon become loose and are then retained by the tongue. A loose-fitting denture predisposes to gingival hyperplasia which will further worsen the fit. Bifid spoons, however, are relatively stable and well-retained appliances, so that the danger of hyperplasia is markedly less. The design of the bifid spoon is such that the torus area is left uncovered and therefore is no hazard to the success of the denture.



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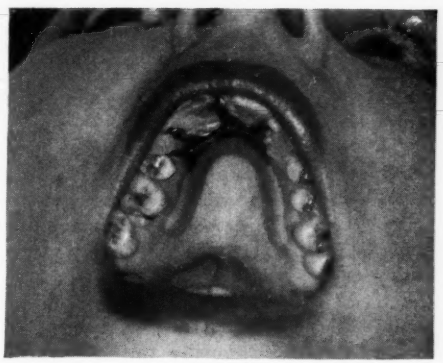

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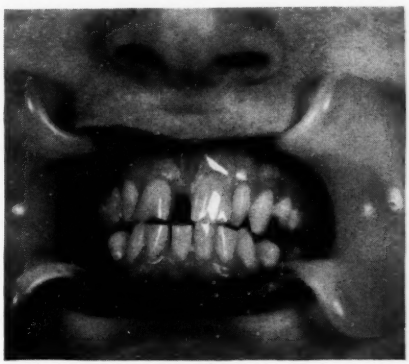

C

Fig. 3.—A, Denture to replace 3|1 and maintain a central diastema. White gold saddle has been used; B, Palatal view of denture. Note distal extension and gingival clearance; C, Frontal view of denture showing wide diastema.

SURVEY OF CASES

Thirty-four cases were undertaken, of which seven were immediate insertions and four immediate replacements; that is, patients were fitted with a denture within four days of the extractions. Twenty of the cases had previously worn dentures.

The number of male and female patients was equal. The average age of the females was approximately 24 years 5 months, the oldest being 48 years old and the youngest 13 years old. The average age of the males was 25 years 2 months, the oldest being 41 years and the youngest 17 years old. These patients came from all walks of life and include a doctor, a lawyer, a school-teacher, a midwife, a professional singer, medical and dental students, painters, fitters, clerks, secretaries, and housewives. Of the dentures constructed, replacing 49 teeth in all, 20 were single-tooth partials, 13 carried 2 teeth, and one denture carried 3 teeth. Five cases have cast-metal saddles and 3 of these carried 2 saddles; one denture replacing 3|3, another 3|1, and the third 4|1. The latter had a large midline diastema.

Twenty-six patients attended for inspection and answered a questionnaire. Three wrote to say their dentures were satisfactory but did not answer the questionnaire. Five patients did not attend for inspections or reply to the questionnaire; one of them had returned to Africa. The average period these dentures have been worn is 10·3 months; the longest 2 years and the shortest 3 months. Of the 26

people questioned, 25 were able to forget they were wearing a denture, and said it was absolutely stable when eating. Twenty-four patients stated the appearance was good and that the denture had not affected their sense of taste; that it was satisfactory to bite on and when doing so stayed firmly in place. Twenty-three said the fit was good, and the denture had no adverse effect on speech or gave any trouble at any time in 22 cases. Sixteen of the 18 patients who had worn dentures before said the bifid spoon was better. Eleven patients wore their dentures at night, although they had been advised not to do so. Two patients complained that during a bout of sneezing their dentures were displaced by the third or fourth sneeze.

Clinical investigation revealed: one case showing caries of the abutment teeth adjacent to the denture, and another with looseness of the abutment teeth. The latter case, when referred to us from the periodontal clinic, exhibited some looseness and pocketing around all the maxillary teeth. A substantial improvement has been made by gingivectomy and a new denture. Two cases complained of soreness of the palate initially, which was due to the bilge keels having been cut in too deeply. This was eradicated within the first week of wearing and no further trouble was experienced.

Palatal mucosa was in good condition, furrowing due to the bilge keels was minimal, and no ulceration was detected. No cases of palatal hyperplasia were encountered other than those that were present prior to the fitting of the bifid spoons and attributable to former ill-fitted dentures. In these cases the hyperplasia seemed to have regressed to varying degrees since fitting the present dentures.

The condition of the gingivæ in all cases was good, no abnormal pocketing was detectable with a probe around the abutments, and there was no mobility of teeth except in the case previously mentioned. The edentulous ridges showed no demonstrable resorption, except in immediate insertion or immediate replacement cases.

Remakes of the immediate cases was generally required from 6 weeks to 2 months after insertion, although two cases that were fitted 18 months ago are still quite satisfactory. Loss of fit due to resorption and remodelling of the ridge results in loss of retention when eating and biting, by sinking of the saddle and loss of fit of the labial flange. The fit and retention of the dentures were good in all but 2 cases.

Appearance satisfied æsthetic requirements in all cases. Four dentures have been broken, 2 due to blows in the mouth. The first occurred when the patient was hit in the mouth by a cricket ball and the second by an elbow when swimming. Of the two others, one was dropped, but the other broke quite unaccountably in the mouth whilst the wearer was eating.

SUMMARY

A variation of the spoon denture is described with details of the clinical and laboratory technique.

Thirty-four cases were undertaken using this type of appliance, and the results of an initial survey are given. All cases except one were successfully treated, this assumption being based on the evidence of clinical investigation and answers to a questionnaire.

Experience has shown that the Kennedy Class IV upper partial denture bearing one or two teeth, occasionally three, may most satisfactorily be constructed by this method. It avoids gingival and periodontal complications and dispenses with direct retainers, so often difficult to design in view of the lack of space between the marginal ridges of the molars.

Acknowledgements.—The author wishes to thank Professor A. S. Prophet and Mr. A. E. Everett for much help and advice in preparing this paper, also Mr. A. C. Lees of the Photographic Department for the illustrations, and the technicians of University College Hospital Medical School for their technical assistance.

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PERFORATION OF A MANDIBULAR MOLAR ROOT BY THE INFERIOR DENTAL NERVE

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SEVERAL techniques have been advocated to minimize complications following the removal of a mandibular molar tooth whose roots are perforated by the inferior dental nerve.

Cogswell (1942), describing the radiographic features which may be present when a root is perforated by the mandibular nerve, advocates the mesiodistal division of the tooth using a cross-cut fissure-bur, thus enabling the tooth to be extracted without dividing the nerve.

Ward (1955) recommends that the nerve be divided with a pair of scissors and the nerve ends be placed in the floor of the socket following the extraction. Sealey (1949) reports a case where the nerve was severed by avulsion in a deliberate attempt to minimize the post-operative hæmorrhage. The degree of anæsthesia following this extraction had considerably diminished four months later.

Parfitt (1921) records the perforation of the roots of a second lower molar. In this case "pins and needles" are reported some four and a half months after the extraction. Sheridan (1941) reports sensation to be normal ten months after the removal of a buried lower right wisdom tooth. Macalister (1958) describes a case in which the nerve was cut with a dental bur during the removal of a perforated molar root.

CASE REPORT

On Sept. 10, 1955, Mr. A, aged 38 years, was referred for the completion of the extraction of his left lower third molar.

Earlier on the same day he had attended his own dental practitioner requesting the removal of this highly carious tooth. In order to facilitate the extraction of the $\overline{|8}$, which was impacted under the distal of the second molar, the patient was advised to have both $\overline{|78}$ extracted, to which he agreed.

Accordingly, under a local infiltration, the $\overline{|7}$ was extracted, but the application of forceps to the $\overline{|8}$ only succeeded in loosening the tooth and, despite the removal of surrounding bone with a dental bur, the tooth although loose remained in the socket.

On arrival at hospital examination revealed a carious and loose $\overline{|8}$ which was painful when touched. The second molar socket contained a cotton-wool pack.

Using a 2 per cent solution of xylotox with 1 : 80,000 adrenaline, anæsthesia was obtained by giving an inferior dental nerve block and a long buccal nerve infiltration in the prescribed manner. Brief application of forceps to the $\overline{|8}$ demonstrated that a surgical technique was required. A buccal flap was raised and bone removed from the buccal and distal aspects using a large fissure bur. Elevators were applied and the $\overline{|8}$ rose out of the socket. The patient at this point complained of severe pain radiating up the left side of his neck and face. Upon removal of the elevator the tooth subsided into its socket. Elevation of the tooth for a second time again produced this severe pain.

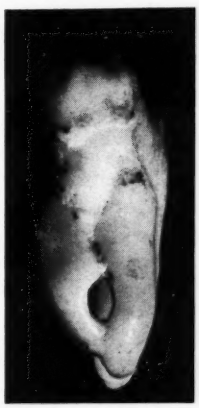

Fig. 1.—Distal aspect of extracted $\overline{|8}$ showing perforation of root through which the inferior dental nerve passed.

At this stage a scalpel was passed down the distal root to the base of the socket. Following this the tooth was safely delivered with forceps.

Examination of the extracted tooth showed a hole through one of its two roots, through which the inferior dental nerve had passed (*Fig.* 1). The two ends of the severed nerve were seen to be lying at the bottom of the socket. The flap was sutured in position and a firm blood-clot was rapidly formed as the hæmorrhage was no more than is experienced in the normal course of events.

The patient was informed that anæsthesia of unknown duration would follow and that if sensation did return its degree might be minimal.

Five days later the patient reported for the removal of sutures. The immediate post-operative period had

proved to be uneventful apart, of course, from the anæsthesia of the lip. Examination, however, revealed both sockets to be "dry"; these being painless, no treatment, other than irrigation, was instituted. Five weeks later the sockets had healed and the patient was experiencing "pins and needles" in his lower lip in the region supplied by the left mandibular nerve.

On April 8, 1958, that is 31 months after the extraction, the patient was again examined. He reported that for the first two months he had experienced "pins and needles" in the lower lip and had then lost all sensation. The only inconvenience he experienced was that occasionally he drank his beverages too hot. He had ceased to worry about the loss of sensation and was only aware of its absence when thinking about it.

Examination revealed anæsthesia of the $\overline{|2345}$ and there was no sensation in the area supplied by the mental nerve.

Acknowledgements.—I am indebted to Professor F. C. Wilkinson for permission to publish this case and to the Photographic Department of the Eastman Dental Hospital for the photograph.

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A SIMPLE TEMPORARY CROWN

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A TEMPORARY crown for use following jacket crown preparation should satisfy two main requirements: (1) A reasonable æsthetic appearance; (2) The protection of the exposed dentine by a suitable material.

The crown to be described provides these two objects with the minimum expenditure of chair-side time, as much of the work may be done by the chair-side assistant.

CONSTRUCTION

A suitable crown form of celluloid or cellulose acetate is first trimmed for close adaptation to the labial shoulder of the preparation. Accurate trimming on the palatal aspect is usually difficult, but should be achieved to the closest limit of the selected crown form.

A silicate cement, which is one shade darker than that of the adjacent teeth, is then selected. After mixing in the usual way, a thin facing of the silicate cement is placed in the crown form as in *Fig.* 1 A. This is allowed to set out of the mouth and the "faced" crown form then filled with a quick-setting zinc oxide–eugenol paste. The whole is then seated on to the prepared tooth. Little excess paste will be extruded labially if the initial trimming has been accurate. Palatally the excess may be pressed with a flat-ended plastic instrument to the correct contour, the final result being as shown in *Fig.* 1 B.

At point X the crown form will often be short of the shoulder, but contouring of the zinc oxide paste in that region will prevent encroachment of the preparation by the gingiva.

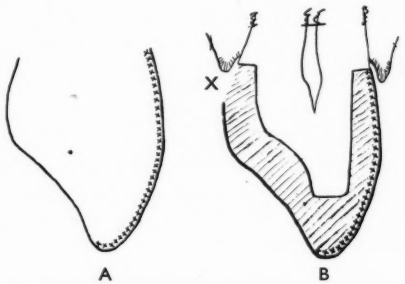

Fig. 1.—A, Diagram showing trimmed crown form with shaded area representing silicate facing. B, Diagram showing completed temporary crown with shaded area representing silicate facing and striped area representing zinc oxide paste.

The reflection of light from the zinc oxide through the darker shade of silicate cement will now provide the correct shade.

THE SYSTEMIC AND LOCAL BACKGROUND OF PERIODONTAL DISEASE*

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INTRODUCTION

THE concept of ever-present systemic and local factors in the aetiology of periodontal disease is an obvious restatement of a biological truism. It is a well-known medical axiom that in morbid states of the body the general features of any disease due to an infective agent will reflect a dynamic balance between the interplay of the "seed and the soil". In the field of periodontal lesions, these ideas have more recently been epitomized by Glickman (1946, 1949) under the brief title of the "Bone Factor". I must straightaway add that, throughout this address, when speaking of "periodontal disease" I include all stages of the condition from established gingivitis through to pocket formation and tooth loss.

By some investigators it has been thought that this so-called "bone factor" applies only to those cases designated "periodontosis" or P. complex—which show certain atypical features both in their history and local lesion. Others maintain that in all examples of periodontal disease, no matter of what particular type, there is a background modified by the constitutional status of the patient. Others again place greater emphasis upon the possible effects upon the gums and teeth of systemic disease elsewhere in the body. It is the purpose of this paper to consider some of the aspects of periodontal lesions in the light of one's modest experience of skeletal disease generally, and to indicate (where this may be feasible) the manner in which the systemic background modifies the local reaction, and its bearing upon the aetiology of lesions of the periodontal structures.

From a very limited study of the vast and rather nebulous literature relating to periodontal disease, it seems to me that there has been much confusion, both in direction and thought, about this constitutional or somatic background—primarily due to a lack of appreciation of the wide variation in the potential response of biological systems (even within one species); and secondly in the lack of uniformity in the nomenclature, classification, recording, and analysis of data relating to the incidence and severity of the disease. In respect of these several aspects, it is admittedly difficult, and in many instances impossible, to decide precisely where to draw a line dividing the physiological and the pathological, e.g., a tissue reaction which may well be a normal variant at one age or in a given environment may, in other circumstances, be indicative of some subtle pathology. Moreover, the mode and extent of the changes observed in any given tissue or system in response to some standardized stimulus will undoubtedly vary considerably between races, species, ages, and sites—just to mention a few of the variable components of the systemic background.

In respect of periodontal disease itself, one has encountered publications describing, for example, gingivitis, which by some writers is reported as being so prevalent in certain population samples as to arouse the suspicion that the lesions described may in certain persons represent little more than an adaptation of the oral tissues to their environment. Gingivitis may be compared here perhaps with other superficial lesions such as sunburn, which in blond nordic types will be experienced after a very brief exposure to the sun. The early stages of this epidermal reaction are clearly reversible; but in some people (with a minimal dose of sunshine) there may appear more serious and lasting signs, such as erythema, blistering, increased pigmentation, exfoliative dermatitis, or even skin cancer. Cognizance may well be taken of the preliminary mild phases, but are we to

* Read at the meeting of the British Society of Periodontology held at the University of Bristol Dental Hospital on May 23, 1959.

give them the full rank and status of a disease? I think that a normal reaction to a known physical agent may well be a better description for the minor reversible degrees of both sunburn and gingivitis.

Similar remarks might be applied to a number of other symptoms which appear in the complicated pattern of periodontal disease, e.g., some reduction in the amount of alveolar bone with limited movement of teeth, minor irregularities in the morphology, and disposition of the gingival mucosa, etc. To my mind there is much to commend the viewpoint that such small modifications in structure and function merely represent an adjustment of the dental apparatus of the individual to the environment, conditioned by circumstances short of actual disease. These thoughts then lead one to adopt a cautious and critical attitude, at least to the literature appertaining to gingivitis, and to some of the surveys relating to periodontitis, especially where the latter have been based solely upon a radiographic examination of the teeth and jaws.

When digesting Parfitt's data (1957) upon distribution of gingivitis amongst children and adolescents, one was struck by the reported chronological relationship of the maximum incidence to the time of puberty in both sexes. Bearing in mind again the time relationship between the eruption of the permanent dentition and puberty, one would conclude that there is some hormonal factor which conditions the appearance of gingivitis. However, it must also be added that the positive correlation between bone growth and puberty is much higher than that of dental eruption; in other words, statistical data here indicate that the development and eruption of the teeth is not so closely under hormonal control as is the case with the main growth of the bony skeleton (Tanner, 1955). Nevertheless, in my short span of life as a histologist I have seen two common conditions formerly considered to be infective in origin resolved largely in terms of hormonal dysfunction, i.e., chronic mastitis and endometritis; although it must be admitted that both these latter diseases are sited in regions not quite so vulnerable to the assail of external agents.

I feel it would be profitable to explore more fully the relationship between gingivitis and the dramatic endocrine changes which occur at the time of puberty. In order to do this one would need to collect information relating to the growth and development of the individual, an assessment of bone and dental age, some measure of the constitutional type (somatotype), an evaluation of the gynandrism, the maleness or femaleness of the individual, and notes of the time of appearance of the secondary sex characters which reveal the phenomenon of puberty. All this is not so complex as it may sound, and careful longitudinal studies along these lines would give more valuable information about "the soil", i.e., the biological background, than would sporadic investigation of some particular biochemical or clinical feature of gingivitis. One would here emphasize the greater likelihood of useful information being obtained from a longitudinal study of selected children than from any number of cross-sectional surveys of population samples. It is timely to recall that a recent study of this type amongst Edinburgh children carried out by Mansbridge (1958) has indicated that there is a positive correlation between sexual maturity and susceptibility to dental caries.

In attempting to appraise the incidence and severity of periodontal disease, many authors have stressed the practical problems encountered in examining groups of patients, the incompatibility of many of the published observations, and the conflicting conclusions. Apart from personal observational errors and natural human bias, these discrepancies have been magnified by the differing techniques applied by many enthusiastic but apparently prejudiced investigators, and have been distorted by too restricted a vista of the biological background and undue preoccupation with some special aspect of periodontal disease. Nevertheless, I believe there are four main points on which there is universal agreement:—

1. Periodontal disease is very common in man, and is a major cause of loss of teeth.

2. The disease frequently appears soon after puberty in both sexes.

3. Periodontitis with pocket formation is progressive with advancing age, but is also relatively painless.

4. That the disease incidence varies greatly from one survey to another, according to the methods of examination and scoring cases, the population and age-group sampled, the date of the study, and the angle of dental disease in which the investigator is primarily interested.

These preliminary remarks serve to emphasize the intricacy of the problem involved in any essay to expound lucidly the part played by the systemic and local factors in the causation and course of periodontal disease. Much has already been written implying that a major contributory factor in periodontal disease is the state of the alveolar bone. This may well be so, but nevertheless I would quarrel with the choice of the term "bone factor". This name I believe to be unfortunate since it will tend to focus attention upon the bone itself, which can be examined all too readily by means of the ubiquitous radiograph, whilst the more significant formative and resorptive soft tissues are likely to be ignored.

Bone itself is a mineralized end-product of an orderly sequence of dynamic metabolic processes in connective tissue, and in most sites has but little bearing upon morbid processes in adjacent soft tissues, save for the physical support given, and by virtue of its function as a reservoir for certain inorganic ions. In connexion with bone one sometimes hears used the term "compensatory hyperplasia" to denote new bone formation as a result of certain abnormal stimuli, e.g., the incompletely immobilized fracture, or the buttressing of axial deformity. I deplore strongly the use of the adjective "compensatory" in this context.

In my view, the tissues seldom, if ever, show reactions to any noxious stimulus which merit the term "compensatory", since this implies an appreciation of damage, dysfunction, or change, coupled with a purposeful attempt to make it good. The more correct viewpoint is expressed in the term "homeotasis", meaning that there are intrinsic mechanisms whereby the mammalian body can adjust itself to function satisfactorily within wide variation of its environment, e.g., maintenance of body temperature, hormone balance and rhythm, etc. This concept of homeostatic controls one may amplify by the added knowledge that increased usage may lead to hyperplasia and/or hypertrophy, whilst disuse tends towards atrophy. One may assume that there are homeostatic mechanisms (under genetic and other influences) which will maintain a healthy mouth and teeth with considerable latitude in race, diet, climate, etc. One may generalize further by adding that function will promote healthy growth and structure of mineralized tissues, whilst disuse will cause dissolution of bone with degeneration and consequent disrepair. I have no doubt that these remarks apply with equal force to the teeth as well as to the rest of the skeleton.

Before passing on to a brief consideration of the tissues of the jaws let us try to catch a glimpse of the complexity of the systemic factor by listing some of its contributory aspects:—

1. Genetic: species, race, family, constitutional type.
2. Environment: climate, water and food supply, geographical location, economic, social, and occupational factors.
3. Personal habits: hygienic or otherwise.
4. Sex (or the lack of it).
5. Growth, maturation, and stature.
6. Mental development.
7. Age: chronological, dental, bone, hormonal, etc.
8. Trauma and repair.
9. Disuse and degeneration.
10. Intercurrent diseases and their treatment.
11. Allergy and hypersensitivity.

The sum total of these many features adds up to the systemic background, with the local or "bone factor" presenting as an inquisitive offspring begot by restricted vision out of curiosity.

Before attempting to discuss the likely bearing of several of these many aetiological factors upon periodontal disease, may I interpolate a section on the morphology and growth of bone and its adnexæ.

ANATOMY, STRUCTURE, AND FUNCTION

At an anatomical level the jaw bones are of much interest. The maxilla grows entirely as a membrane bone, increasing in size by ossification at four sutures, and in thickness by periosteal appositional growth. The major part of the mandible also is formed in membrane, but there is a segment of the ascending ramus and a sliver of the coronoid and symphysis menti which are of endochondral origin. These growth peculiarities have a bearing upon the range of tumours that arise in the jaw bones, e.g., giant-cell tumour is not uncommon in the mandible, but extremely rare in the maxilla. All the alveolar bone, of both jaws, is membrane bone, i.e., grows in condensed fibrous tissue; it thus resembles in mode of growth the membrane bones of the calvarium, and there is also structural similarity. The general form consists of two outer boundary plates of lamellar bone, within which lies a fine trabecular network of cancellous bone. This osseous pattern may be contrasted with the compact corticalis of the long bone shaft, whose structure is modified by a genetically conditioned process of endochondral ossification qualified by a more strenuous function.

So far as the actual bone of the human jaw is concerned, I have no knowledge of any fundamental differences in morphology or metabolism as compared with cancellous bone elsewhere. The bone present in mandible and maxilla may be coarse or finely textured fibre-bone, or lamellar bone, according to its site. Cementum again is a modified form of bone, differing morphologically only in its tendency to become acellular. Other departures from usual bone structure and metabolism may well exist in cementum, but so far as I am aware, they have yet to be demonstrated.

Let us turn for a moment to compare the periodontal membrane with the periosteum (*Fig.* 1 A, B). These are both soft tissue layers which contain cells having formative and destructive functions in the growth, maturation, and repair of the related mineralized tissues. Morphologically periosteum consists of an outer fibrous layer and an inner cellular layer which is potentially osteogenic—sometimes even chondrogenic (as also may be periodontal membrane). There are also scattered areas of bone resorption (Howship's lacunæ) where may be found multinucleated giant cells or osteoclasts (*Fig.* 1 D). These cell types all occur in periodontal membrane, but whereas periosteum is an investing layer, clothing the external surface of bone, periodontal membrane lies between the cancellous alveolar bone and the radicular cementum. As with bone, the mineralized cementum is preceded by a collagenous matrix or cementoid which is laid down in an orderly manner by cementoblasts—cells which are morphologically identical with osteoblasts (*Fig.* 1 B). The first, and possibly the most important difference between these two investing and formative layers lies in this fact that the periodontal membrane occupies a very confined space in the tooth socket, being bounded upon one side by alveolar bone and on the other by cementum. In my very limited experience of the histological structure of periodontal membrane, it appears to be relatively poorly supplied with blood-vessels as compared with periosteum.

In periosteum, especially that of the major appendicular long bones, trauma and/or septic infection initiates the classical sequence of tissue reaction: (1) Hyperæmia; (2) Exudation; (3) Leucocytosis; (4) Proliferation.

The histologist usually sees the changes from the exudative stage onwards in material examined; but in a specimen of sufficient size one may observe all four phases as one passes from the septic or damaged area into the adjacent tissue.

In periosteum the second stage leads to swelling—sometimes up to a thickness of 1 cm. or even more. This reaction is clearly impossible in the restricted space of the tooth socket, although no doubt some degree of it may be seen in the gingivitis which may accompany or precede the formation of a periodontal

pocket. This swelling of the periosteum, if of any chronicity, is almost invariably followed by some amount of new bone formation to which there may be several contributory factors:—

1. Increased blood-supply.
2. Cell proliferation and differentiation to osteoblasts.
3. Spatial separation of the osteogenic deeper layer of the periosteum from the normally subjacent mature bone.
4. The stimulating effect of metabolites.

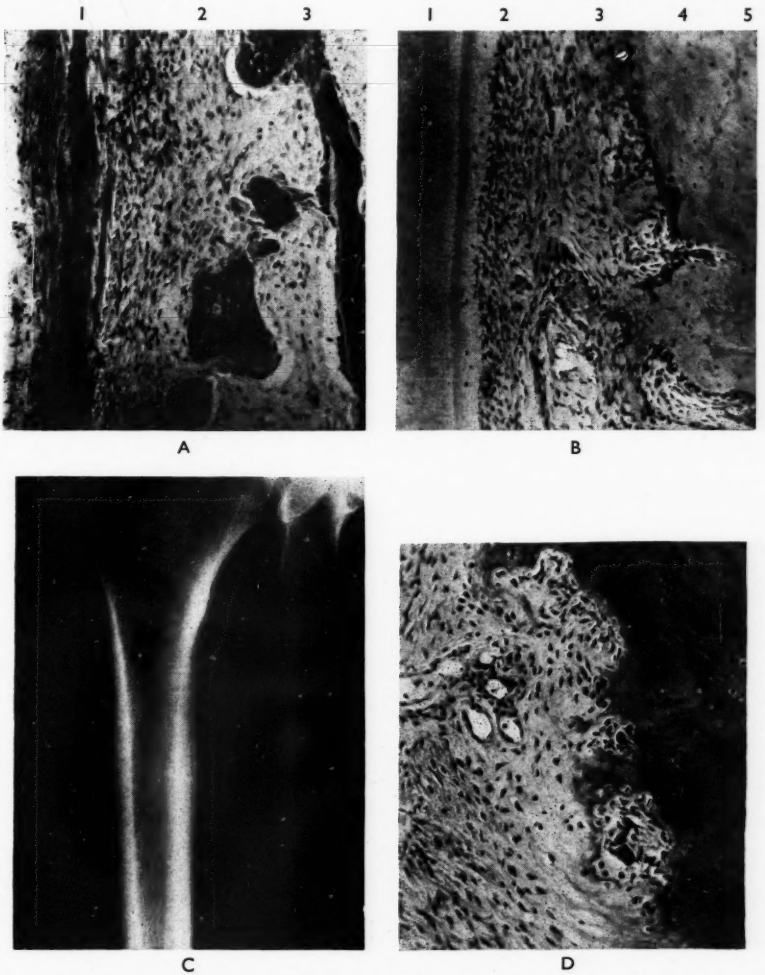


Fig. 1.—A, Periosteum (rib of dog). 1, Fibrous investing layer; 2, Cellular osteogenic layer; 3, New fibre-bone, margined with plump osteoblasts. Hæmatoxylin, phloxin, and tartrazine. (× 108.) B, Periodontium (human). 1, Dentine; 2, Cementum, with mantle layer of cementoblasts; 3, Fibrocellular layer; 4, Osteoblasts; 5, Fibre-bone of alveolus. H. and E. (× 108.) C, Focal inflammatory periostitis of femur (BTR/289, F. aged 59). This was a painful condition of several years' duration. Note irregular new bone formation in region of lesser trochanter and medial border of upper part of the shaft. D, Periosteum of tibia (BTR/692, M. aged 5). Howship's lacunæ showing several osteoclasts. Note the eroded margin of the bone which is here undergoing re-modelling. H. and E. (× 108.)

In some instances where there has been an indolent smouldering periostitis, the amount of new bone formation may be enough to arouse suspicions of the presence of a bone-forming neoplasm. This newly-deposited periosteal bone is usually laid down in one of three characteristic patterns, or in a combination of these: (1) Lamellar (so-called "onion-peeling"); (2) Spicular or sun-ray; (3) Irregular (*Fig.* 1 C).

But in each instance it is fair to say that the pattern of the new bone so formed is more typical of the *type* of bone involved, e.g., long, flat, irregular, etc., than it is of any specific lesion. The bone pattern of a reactive periostitis may be perfectly mimicked by the widespread effects of a primary or secondary neoplasm. Moreover, the extent of this new bone formation will be modified according to its anatomical site, the age of the patient, and the nature and duration of the disease process; furthermore, it may be more pronounced in some species than in others, e.g., dogs.

The chain of events seen in the periosteal reaction called Hypertrophic Pulmonary Osteoarthropathy or Marie's disease is probably rather similar in its nature (*Fig.* 2). This bone reaction is usually, but not invariably, associated with some intrathoracic lesion—pulmonary T.B. or neoplasia. At present, all available evidence suggests that in this condition the periosteal reaction to the remote cause is mediated by some interference with the sympathetic nervous system, and so with the blood-supply and capillary circulation of the periosteum (Holmes and Price, 1958). These proliferative phenomena so characteristic of long and tubular bone periosteum are not, I believe, seen in infective states of the periodontal membrane; although in chronic infections some small amount of new reactive bone or cementum may uncommonly be observed. Increase in cementum seems to be more a feature of advancing age than it is of chronic periodontal disease.

One would suggest then that this difference in the reactivity of these two comparable investing tissues is basically related to the inability of the periodontal membrane to swell much, save at the expense of the adjacent bone or dental structures.

Pondering the clinical details of a number of incidence surveys of periodontal disease, one is struck by the fact that many persons must live quite happily with their periodontitis until they become almost edentulous, or are warned by some enterprising periodontist of the dire consequences of their dental neglect. This to me suggests that the disease is for the many a relatively painless condition; although from personal experiences "in the chair" I have no doubt that there is a good supply of exquisitely sensitive nerve-endings around the tooth root as well as in the pulp canal.

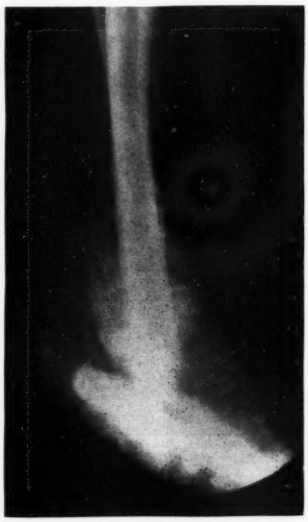


Fig. 2.—Hypertrophic pulmonary osteoarthropathy (Marie's disease). X ray of lower rear limb and ankle of a dog whose lungs were involved by metastatic carcinoma. Note the extreme degree of periosteal new bone formation, which in this instance has proceeded to osteophytosis.

Periostitis is, by comparison, a very painful condition, and the bone reaction of hypertrophic pulmonary osteoarthropathy may be the cause of severe and intractable pain, bad enough even to warrant amputation of a limb or a severe operation to essay the removal of an intrathoracic tumour mass. This difference may well be due to the marked swelling of

periosteum which does not occur in periodontal membrane. Moreover, although the latter may undergo some small increase in tissue pressure, there is evidence to suggest that it is the tension and stretching of nerve-fibres rather than direct pressure which is responsible for peripheral (as distinct from nerve-root) pain.

With regard to the effects of pressure upon bone generally, one has already emphasized that normal pressure, e.g., weight-bearing or mastication, will promote healthy tissues. Increase of pressure, however, beyond the normal range may lead to rapid formation of new bone (as shown by Charnley, 1953; and by Friedenburg and French, 1952). It is not at all certain yet if increase in pressure upon bone does eventually favour excessive bone formation; in fact, such evidence as there is rather indicates the reverse as being more likely. This is shown by the bone resorption associated with the sustained pressure of an adjoining aneurysm or benign tumour. Hancox (1956) expresses the view that mechanical pressure leads to bone resorption by the activity of osteoclasts, as also, of course, does injury, parathormone and certain substances of known chemical nature, e.g., vitamin A. It is, of course, also the classical view that it is increased pressure which initiates absorption of the periradicular bone in the shedding of the deciduous teeth.

It seems likely, then, that there is some increase of tissue pressure or tension in the septic socket, but associated with bone dissolution rather than bone formation. These assertions are supported by the results of orthodontic manœuvres which show that alveolar bone is resorbed upon the pressure side of a tooth being moved, and some new bone laid down upon the tension side. By analogy with this latter finding one may consider the bony spurs arising in long bones which one may sometimes see caused by the prolonged overaction of some major muscle group, e.g., adductor longus in equestrians. In terms of anatomical growth, the major tendons and fascial septæ influence the development of bony tubercles and raised ridges, e.g., tibial tubercle, and linea aspera of the femur.

Periosteum is usually described as consisting of two ill-defined layers—an inner cellular or osteogenic layer, and an outer collagenous fibre layer, in which the fibres are oriented parallel with the long axis of the bone. The inner cellular layer may not always be obvious, but the potential remains for its rapid development.

These longitudinal fibres are mainly absent in the periodontal membrane, in which the fibres tend to run radially from the alveolar bone to cementum. This is an arrangement more like a tendinous insertion into a long bone, and the fibres serve the same purpose, i.e., transmission of tension. These tendinous insertions have a tendency throughout life to undergo fibro-chondroid metaplasia, suggesting that the cells of tendon are more closely allied to fibro-cartilage cells than is the case of fibroblasts elsewhere. For some unknown reason fibro-cartilage does not seem to have quite the same avidity for calcium salts as does hyaline cartilage, particularly that found in the cartilages of the respiratory skeleton and the ribs. This difference may possibly be due to movement and transmission of tension by tendon fibres, with some repeated spatial disturbance at a molecular level, so counteracting the tendency to calcification with advancing age. It seems possible that these features also apply to the analogous structures of the periodontal membrane. A rather similar conclusion may be reached by a different line of reasoning.

One may regard the tooth in its socket as a kind of joint, a type of syndesmosis in which bony surfaces are opposed but united by unmineralized collagen fibres. The other good example of this type of joint is the sutural articulation between adjacent bones of the calvarium. According to *Gray's Anatomy* (p. 274) the skull sutures commence to ossify at about 30 years of age, and this continues until eventually there is complete bony union as a normal feature. At these sutures during adult life of course there is little or no movement; where slight movement occurs in a simple joint of this type cartilage develops, as in a symphysis or a poorly-controlled fracture. In fact, Orban (1928) was able to

demonstrate that cartilage may even appear in the periodontal membrane in conditions of unusual or modified stress. Bony ankylosis of teeth is, I believe, relatively uncommon—obviously it is not a feature of periodontitis—where bone loss is the rule. Presumably, in the normal tooth socket the tension transmitted by the regular mastication interferes with the physico-chemical environment which is a pre-requisite for mineralization. There are also other subtle changes necessary—probably in the interfibre cement substance—before ossification can occur, but one may conclude that the process is not favoured by tension in the local collagen fibres.

There is also some evidence to indicate that the type of bone which does form from fibrocartilage (bundle bone), and is related thus to tendinous or perforating fibres, is more liable to demineralization and dissolution than is the mature compact lamellar bone of cortical situation. This may often be well observed in cases of hyperparathyroidism.

Having thus compared and contrasted the formative tissues of periodontal membrane and periosteum, let us now consider the normal mechanism concerned with bone devolution and removal.

We are perhaps on much less certain ground. It is widely accepted that the osteoblast forms bone, but there is by no means universal accord as to the precise role of the multinucleated giant cell, the osteoclast. In the appendicular long bones of endochondral growth, a good deal of remodelling takes place (as also in the ascending ramus of the mandible). This occurs by gradual resorption of bone both within and without, and the accrued evidence suggests that in this the osteoclast takes part. McLean (1956) expresses the present view in a choice pearl of understatement: "An exhaustive review by Hancox (1949) concludes that there is no direct evidence that osteoclasts erode bone, but that their constant occurrence in zones where resorption is taking place is more than incidental."

These large amœboid motile cells are to be found wherever bone growth and remodelling occur—in the metaphysis of the long bone, around islets of unresolved cartilage matrix in the primary spongiosa, etc. They are also very frequent in disease of bone where resorption is active, e.g., Paget's disease and osteitis fibrosa cystica. Moreover, these cells are common around the deciduous tooth

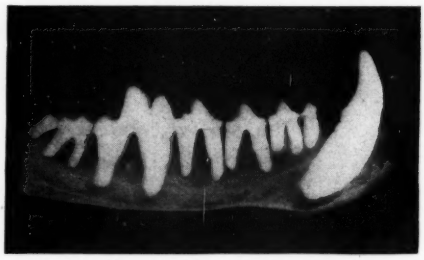


Fig. 3.—X ray of mandible of 9-year-old terrier dog with "rubber-jaw" syndrome—secondary hyperparathyroidism, following chronic nephritis. The whole bone was involved by osteitis fibrosa with absorption of the periradicular bone, but no loss of teeth.

radicles, so much so in fact that sections of jaw bones have often been used as subjects for illustrations of osteoclasts (Arey, 1919).

It has become increasingly clear that the osteoclast appears as a result of some unknown chemotaxic stimulus from bone, which is then eroded, leaving the "nibbled" appearance of the typical Howship's lacuna, or the so-called "festooning" of Paget's disease. How this is brought about is not known; but I think that quantitatively the amount of osteoclastic activity may vary according to the type of the bone, and the site of the destructive lesion in it. Bone destruction may also be due to other cells, macrophages and some tumour cells.

From first-hand knowledge I have but little experience of osteolysis in the jaw bones, but have no good reason to suppose that it differs markedly from the response of other bones elsewhere. In the condition sometimes seen in dogs called "rubber-jaw", which is a secondary hyperparathyroidism following chronic renal damage, there is extensive osteoclastosis of the mandible producing a flexible dystrophic bone. This is associated with a considerable amount of absorption of periradicular alveolar bone (*Fig.* 3)—although there may be no loss of teeth. The long bones do not appear to suffer in this disease

nearly as much, but one may detect early changes in the ends of the bones, beneath the periosteum, and also in the tufts of the terminal digits (Holmes, 1957). In both of these sites there is some amount of irregular bundle bone

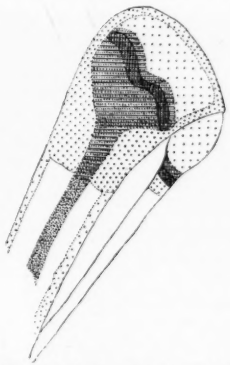


Fig. 4.—Total acid phosphatase activity of upper end of tibia and fibula of rabbit (B4), aged 5 months. (The enzyme activity was estimated at *p*H 4·9, and is proportional to the depth of spotting and shading. The values for acid phosphatase run from less than 1 unit (clear areas) to 40 units per gramme of dried powdered bone. (The unit here used expresses the number of micromoles per hour liberated from paranitrophenyl phosphate per gramme of bone.) It may be noted that the maximum concentrations were found in the region of the epiphysial growth cartilage, and in the medullary cavity. (*By courtesy of Dr. Grace M. Jeffree.*)

connected with perforating fibres. I have never seen such a striking change in a human mandible in any example of hyperparathyroidism, so that this extreme reaction to parathormone in the canine jaw bone must be regarded at present as a species peculiarity.

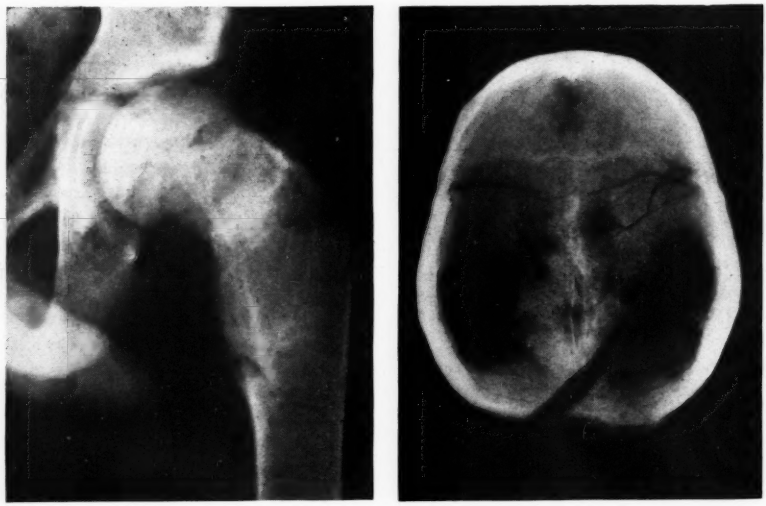
In connexion with bone resorption it is a striking feature how the mandible alters in shape with senility. This age change has recently been studied by Blacharsh, Staub, and Mangolis (1958) in 169 males who were hospital in-patients for a variety of reasons. The series were sub-divided into 3 groups according to whether the patient was suffering from a surgical, medical, or mental complaint. There was no significant difference between the groups, in whom it was shown that alveolar bone loss in the mandible increased as a function of age, rather than as an expression of any particular type of systemic disease. One may add that rather similar bone loss may also occur in the membrane bones of the calvarium, where some thinning is a not uncommon feature in aged persons. This change may, however, become extreme in some individuals where there is a superadded senile osteoporosis present (*see Fig.* 5 B). The major known factors concerned in this atrophy of the parietals are vascular changes, the absence of any tensile effects of muscle-fibres, and the gradual diminution in hormonal activity which accompanies advancing age.

To move now from the histological level to the biochemical in respect of bone growth and removal, it has long been known that ossification is associated with alkaline phosphatase. This is well supported by conventional biochemistry, and by histochemical studies of growing bones, fracture callus, etc. Evidence is now accumulating to suggest that the remoulding and resorption of bone may be related to the activity of the acid phosphatases of bone, and Changus (1957) has recently demonstrated the presence of this enzyme in the osteoclast. Furthermore, Kochakian (1952) has shown that acid phosphatase is increased in amount in bone following parathormone injections.

Dr. Grace Jeffree (1959), working in my laboratory, has recently made a quantitative study of the distribution of acid and alkaline phosphatases in the growing bones of young rabbits. It was of great interest to note in her phosphatase maps of the bones, that the acid phosphatase was mainly demonstrable in the endosteum and growing ends of bone, both sites where bone resorption is known to be active (*Fig.* 4). There is also evidence from Crétin (1951), quoted by Hancox (1956), which suggests that there may be a *p*H gradient around osteoclasts, dropping perhaps as low as *p*H 6·4 for a space of 20 to 30 μ. This more acid reaction of the tissue fluid would tend to enhance acid phosphatase activity, hence to aid bone destruction. It seems likely, therefore, that bacterial infection of a tooth socket with stagnation and enzymic degradation of food accretions could also lead to some fall in the local tissue fluid *p*H, which in turn would again favour osteoclastosis.

Other evidence also suggests that an acid *p*H favours demineralization of bone at least, and that the leaching-out, as it were, of the microcrystalline bone salt is the first stage in devolution of formed bone. There is good reason to suppose that the bone salt exercises a stabilizing effect upon bone, firstly by bonding the macromolecules of the inter-fibre cement substance, secondly by contribution to the general pool of "mobile" calcium, which again I visualize as being a stabilizing (or homeostatic) force by virtue of the fact that calcium forms relatively insoluble compounds with proteins and mucopolysaccharides. Certainly, in the major bones of the body one does not encounter much disease during the middle period of human life, when one may assume that maturity of structure at a molecular level is associated with a relatively low rate of metabolic turnover; the same remarks may of course apply to the relatively inert compact corticalis of the adult long bone shaft.

From what has already been said, one can conceive of processes which may lead to an acid *p*H in a septic pocket or tooth socket, and that these changes are likely to favour bone destruction and tend to spread rather than be self-limiting. By contrast, the marked vascularity and ability to swell of long bone periosteum are such that any focal change in *p*H must be readily adjusted by the more rapid interchange of tissue fluids and removal of metabolites. That such demineralizing changes may also be brought about by chelating agents at a neutral *p*H must also be borne in mind. However, in terms of biophysics the combination of calcium ions with large polyampholytic macromolecules would not to my mind involve a rapid loss of calcium from a bone trabecula by diffusion, such as may take place in the form of the smaller and more mobile chloride and bicarbonate molecules. At present we have but meagre knowledge of any naturally occurring metabolites which may function as chelating agents; but bone loss has been



A B

Fig. 5.—A, Osteomalacia. M., aged 15. Note the two pseudofractures or remodelling zones in the femur ("umbauzonen" of Looser); other typical sites for such lesions are the pubis, scapula, and ribs. B, Osteoporosis (advanced) of parietal bones. F., aged 87. Some thinning of the calvarium in this site is not uncommon in old people—but not to this extent. A histological section made from the posterior block of bone showed almost complete cessation of osteoblast activity.

caused in experimental animals by the injection of chelating agents such as versene (ethylene-diamine-tetracetic acid).

There is one dental feature which is peculiar to the jaws and which has been incriminated by many in the causation of periodontal disease, i.e., the close proximity on the gingival epithelium to bony tissues. In the normal tooth the marginal epithelium is attached to dental enamel or enamel cuticle. Gottlieb (1921) and others have maintained that there is a migration of this junctional line in an apical direction, and that the crevicular epithelium may eventually be attached to cementum. Others again have attempted to demonstrate histologically the re-union of epithelium with cementum in experimental lesions and after certain modes of treatment of periodontal pockets. Now my personal experience of this aspect of periodontology at first hand is nil; but I view these claims with some misgiving. One can well accept the presence of fine keratin fibrils running betwixt epithelium and enamel, itself an epithelial by-product, but I find it difficult to accept that such structures may be incorporated into cementoid, let alone into mineralized cementum. It would be necessary for such a union to take place prior to mineralization of the organic matrix of cementum, and I very much doubt whether any mammal can utilize a protein other than collagen in this fashion. Hence one is chary of accepting allegations of firm fusion of epithelium to cementum or to dentine. Recent experimental work in fact suggests that only collagen among the fibre-proteins, and probably only the naturally occurring collagen with the 640 Å. banding, is able to induce the deposition of hydroxy-apatite microcrystals (Glimcher, Hodge, and Schmitt, 1957). This doubtful ability then of keratin-forming epithelial cells to acquire any firm anchorage to mineralized connective tissue structures based upon collagen fibres is possibly one of the key points in the transition from gingivitis to pocket formation and the downwards proliferation of the periodontal mucosa.

On the other hand, Pinborg (1958) in a study of neoplasms of the dental adnexæ has claimed that organized structures which may be histogenetically derived from the enamel-forming dental epithelium may induce in their immediate vicinity the formation of dentine or cementum. This interesting suggestion is of course analogous to the embryologist's concept of evocators and organizers, compounds which are cell specific and control the tissue differentiation and morphogenesis. If we can accept then that some part of the crevicular epithelium is derived from the enamel organ, such an area may retain the ability to induce the formation of a thin layer of cementoid with which it may be intimately conjoined in some obscure fashion, this cementoid in turn fusing with the pre-existing cementum of the tooth root. We are then perhaps one step forward, but are faced with two fresh problems:—

1. With the effects of disease and advancing age, does the residual crevicular epithelium lose its ancestral capacity to induce this hypothetical cementoid and so perhaps attain re-attachment to the root?

2. Does this type of dentally conditioned epithelium present in the adult socket, or does it vanish with the years, so that the margin and sulcus come to be lined entirely by a *gingival* epithelium?

It is typical of epithelium elsewhere in the body to grow along tissue planes, to cover denuded surfaces, or to invade the submucosa if there is any local growth stimulus present. This may be well seen in the edges of healing ulcers, and in the spread of carcinomata. That this proliferation of gingival epithelium is due to age changes in the tissues is unlikely, since with advancing age one sees most often a generalized atrophy of the epithelial papillæ with thinning of the supra-papillary zone of the mucosa.

From a limited study of this important anatomical region of the periodontium one is impressed by two points:—

1. Medially there is a limiting epithelial edge.

2. The small size of the area of gingival mucosa which is involved in periodontal disease of the unit tooth.

By comparison with the behaviour of epithelium elsewhere, one would well suppose

that if the edge of the crevicular mucosa becomes detached from the enamel from any cause, it would naturally tend to proliferate. This detachment produces what is in effect a small ulcer, i.e., connective tissue denuded of its superficial protective covering layer. Moreover, in view of what takes place in other superficial ulcers elsewhere one may expect to find downward tracking of epithelial cells at some little distance beyond the actual extent of the denuded surface, and not only at the precise edge. One must add that there may be marked proliferation of the skin epidermal layers in inflammatory states of the subjacent dermis, even in the absence of any superficial breach of continuity. It is necessary also to emphasize the bearing of the integrity and health of the sub-epidermal or sub-mucosal fibrous layer upon its covering epithelium. Certain aspects of this inter-dependence have been well described and discussed by the most interesting paper of Mackie and McGovern (1958).* Orban (1952) states: ". . . the proliferation of the end of the epithelial attachment always takes place before the separation of the attachment at the bottom of the sulcus or pocket has reached the end of the epithelial attachment". One wonders to what extent this is entirely true—it certainly raises a number of queries:—

1. Is there in fact a *physiological* migration of the epithelial attachment from enamel to cementum—or is it pathological?

2. If and when such a migration takes place, does the epithelium remain firmly attached *all round the tooth* (to cementum)?

3. If firm union does not so take place, is it surprising that the epithelium proliferates, and that there is bacterial infection with consequent damage to connective tissue fibres, cementum, and bone?

From these ideas one feels rather that the solution to the progression from gingivitis to periodontitis simplex largely lies in the peculiar anatomy of the tooth socket, being also as formerly suggested subject to some hormonal influence and aggravated by infection from without. Other systemic factors would seem to be of relatively small importance in this respect. The vital point here surely must be that continuity of the epithelial layer is essential to protect the subjacent connective tissue. If this layer is harmed beyond repair, then gingivitis will proceed to periodontitis simplex; but if the damage is only small, then the epithelial breach may be healed with no further spread of infection via this portal.

These conclusions are strengthened, I think, by features to which I shall return in more detail later:—

1. In persons over 35 years of age (Russell, 1957) the annual incidence of periodontal disease with pocket formation approaches closely to that of gingivitis.

2. A characteristic feature of advancing age is an inability to repair and make good tissue damage, be it epithelial ulceration or bony fracture.

It is, perhaps, in respect of this feature of the modified response with advancing age that we may discern the systemic effect upon the progress of the preliminary stages of periodontal disease. Finally, one may add that cutaneous ulcers in older persons are notoriously indolent, and respond poorly to any form of treatment.

OSTEOMALACIA AND OSTEOPOROSIS

It is now necessary to speak briefly about the two main forms of bone devolution, osteomalacia and osteoporosis.

By osteomalacia we mean a demineralization of existing bone. This may be caused by inadequate intake of Ca in the diet, by faulty Ca absorption from the gut, by increased Ca loss, or by deviation of Ca to serve pressing bodily needs other than those of the skeleton. Osteomalacia is now a rare condition in Great Britain, although sub-clinical states are still seen from time to time. In the axial and appendicular skeleton, about the earliest signs of this deficiency are the appearance of pseudo-fractures, so-called "Looser's zones" or "umbauzonen". These appear as tiny cortical cracks to be found in the pubis, the femoral neck, and the lateral border of the scapula

* To those who are deeply interested in gingivitis, its causation and course, I feel that much might be learned from a careful perusal of the paper by Mackie and McGovern (1958).

(*Fig.* 5 A). To my mind this rare malady can have but little part to play in periodontal disease, save possibly in the case of pregnant or lactating women, in chronic renal disease, or in those with severe biliary or pancreatic dysfunction.

By osteoporosis, on the other hand, we indicate a condition in which the formation of osteoid and new bone fails to keep pace with their normal removal; hence on balance there is bone loss (*Fig.* 5 B). This may be due to quite a number of causes, the most important of which are: (1) Protein deficiency; (2) Immobilization and disuse; (3) The menopause; (4) Old age and senility; (5) Suprarenal cortical hyperplasia.

In this brief incomplete list we may note conditions which may have some bearing upon periodontitis and periodontosis.

In both the fields of ecology and animal experimentation we have evidence to show that protein deprivation leads to bone loss. This type of dietetic deficiency may possibly account for the very high incidence of periodontitis reported by Marshall-Day and Schourie (1949) amongst Indians; but it is anomalous that a relatively low incidence was observed amongst Egyptians by Dawson (1948), and amongst East Africans by Schwarz (1946), the latter data being considerably less than those found from several surveys in North America by Marshall-Day, Stevens, and Quigley (1955) in Boston, and by Miller and Siedler (1940) in New York.

However, the dystrophic effect of a low protein intake upon bone seems to have been firmly established by Glickman, Morse, and Robinson (1944), and by Chowla and Glickman (1951).

In the appendicular long bones, disuse rapidly leads to osteoporosis, and I have no doubt that the constant and regular usage of the teeth is one of the main factors in maintaining a healthy mouth. (This may be observed amongst the urbanized dogs who suffer extensively from periodontal disease, and who are fed usually in a most unsuitable manner by their emotional owners—often soft food, with an excess of soft cereals, rather than a natural diet of raw meat and bones.) I think it would be fair to generalize to the effect that periodontal disease is less likely to afflict the individual who regularly consumes a liberal and varied diet in which there is sufficient hard and fibrous material to provide ample work for teeth, gums, and masticatory muscles.

Whilst speaking about the role played by the function of the teeth as a means of preserving healthy jaws, I am not prepared to embark upon a detailed discussion of the possible effects of occlusal trauma. Surely, the continued use of the teeth in the adolescent is one of the main factors which regulates their precise location in the jaws. Thinking again of the tooth socket as a very slightly movable joint, one may state that in such joints elsewhere abnormal physical stresses and strains may lead to degenerative changes (osteoarthritis), with damage to the articular cartilage and bone. However, in the majority of cases of advanced osteoarthritis the cause of degeneration lies elsewhere than in some faulty posture or abnormal weight distribution. The more usual view is that one may attribute the joint changes to age changes in the articular surface, although no doubt wear and tear take their toll with the passing years. In short, I think that the homeostatic mechanisms of the tooth socket must be well able to adapt to a good deal of variation in stress and strain before failing, and so permitting the development of defects sufficient to initiate a periodontal lesion.

We now come to post-menopausal and senile osteoporosis, both of which are fairly common and produce extensive skeletal changes. The cause of the former is mainly hormonal, and in some the condition may be halted or alleviated by the administration of œstrogens and testosterone.

Both of these forms of osteoporosis by leading to resorption of alveolar bone probably contribute to the progress of established periodontitis, and may be responsible for some cases of periodontosis in older persons. However, since they both appear in persons aged 50 years or more, when the annual rate of increase of periodontal disease is falling, one is forced to conclude that they play only a minor role in the causation of the majority of cases.

I shall only briefly comment upon the possible bearing of suprarenal cortical hyperplasia. It is well established that the suprarenals take part in the general hormonal crescendo of puberty, increasing in size and output of œstrogens and androgens in both sexes. This is the so-called "adrenarche" and is under pituitary control, but also conditioned by other factors—genetic, nutritional, climatic, etc. We do not yet know with any certainty if there is a concomitant increase in output of corticoids, but changes do occur which could theoretically modify protein metabolism. It is established fully that cortisone is antagonistic to collagen formation, hence to wound healing and tissue repair; this compound when injected will cause osteoporosis, which is also seen in idiopathic suprarenal hyperplasia.

The hormonal story is an extremely complicated one and by no means fully understood, and within the compass of this address it is impossible to do other than indicate just the most obvious relationships between hormonal effects and tissue changes.

That these and other steroid hormones play some part in the aetiology and course of periodontal disease is undeniable; there would seem to be broad clinical observations which indicate this. Their mode of action, however, is obscure and indefinite, as concluded by Shroff (1956).

AGE

Let us now consider more closely the effects of ageing of the body and how this may have a bearing upon periodontal disease.

First of all, what do we mean biologically by ageing of the tissues or senility? Quite briefly, the term "age changes" may be taken to define certain biological trends the summation of which indicates a declining physical, metabolic, and mental activity. Here I do not intend to enlarge upon the biochemical or biophysical changes that are known to occur with advancing age.

Minot wrote in 1889 that "the retardation of growth is old age, and its cessation is death". This, then, implies fairly enough that ageing begins soon after the peak activity of the system or organism is passed (however painful this thought may be to the under 40's) Moreover, it has been suggested that age changes are probably most rapid in their earliest stages, but, of course, not all these changes or their rate of progress coincide in chronological time.

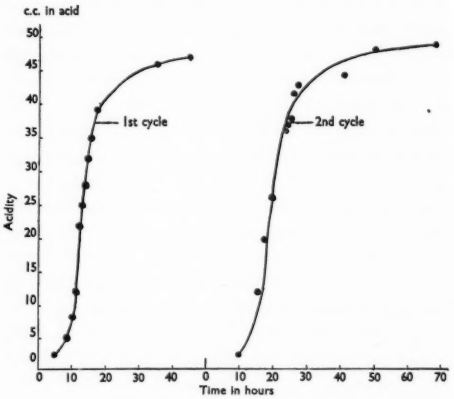


Fig. 6.—The first cycle represents the course of growth and senescence of a culture of lactic-acid producing bacilli in milk. Growth ceases after a characteristic concentration of lactic acid is attained. The proof that the accumulating acid is the limiting factor in growth is furnished by the fact that when the sour milk was neutralized with lime-water, a second growth cycle was produced, virtually a duplicate of the first cycle. Similar curves result when food is the limiting factor. Cessation of growth leads to death of the bacteria. (*Redrawn after Brody, S.* (1945).)

Broadly speaking, advancing age denotes a lowering, of the basal metabolic rate; a diminuendo in hormonal and enzymic activity; a decrease in the cardiovascular-renal reserve; a gradual loss in the lenticular accommodation of the eye; a lessening of muscular strength and endurance; and a vitiation of the ability of the body to repair tissue damage of any form (*Fig.* 7). Where these various features can be measured, e.g., muscular power, and the data plotted against chronological age, the resulting curves, like those of growth, are usually sigmoid in type.

To comprehend the meaning of these curves we turn to organisms of a much simpler type, e.g., bacteria or yeasts (*Fig.* 6). In these, we find that growth, i.e., multiplication, proceeds unchecked, provided that there is an

adequate supply of suitable food, and waste products are steadily eliminated from the environment. If growth ceases, due to non-compliance with these two requirements, then

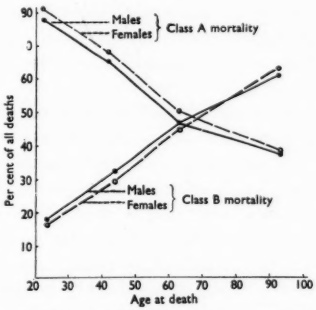


Fig. 7.—Age trends in percentage mortality due to breaking down of (A) organ systems normally in direct contact with the external environment; and (B) organ systems not in direct external contact—plus "senility". *Note*: It should be pointed out that mortality may not truly reflect morbidity; nevertheless, certain broad trends are obvious. (*Redrawn from Brody, S.* (1945) *after Pearl, R., and Raenkham, T.* (1932).)

senescence sets in and death soon follows. By like token, tissue culture work with human and chick fibroblasts indicates that similar laws apply to tissue cells, which are therefore potentially immortal. It is significant that in most mammalian species the total life span is normally about five times the period of growth.

On considering the broad effects of age upon disease, we find that the trends in mortality and morbidity fall into two main classes; these are shown in *Fig.* 8 (Brody, 1945).

Group A.—The curve shows the incidence declining with advancing age. By and large this includes diseases due to organs or systems in direct contact with the external environment and so affected readily by external agents, e.g., the exanthemata, typhoid, etc.

Group B.—Here we see the incidence of disease increasing with age. This group mainly covers organ systems not in direct contact with the external environment, but which are liable to insidious degenerations. Here one may indicate cardiovascular disease, rheumatism, cancer, etc. We shall see in a moment that the age distribution curves of periodontal disease have features suggesting that there is in fact a combination of two curves, one characteristic of Group A, in juveniles, and the latter part of the annual incidence curve fitting better into the general form of Group B (*Fig.* 12). Or, to put it another

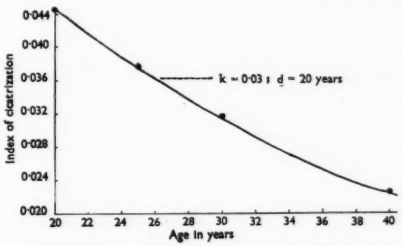


Fig. 8.—The age course of senescence in man as measured by the time rate of healing of wounds at different ages. *k* represents the percentage decline for the unit of time indicated on the age axis—in this instance 3 per cent decline per annum. *d* represents the time taken for any value to decrease by half. The equation fitting this curve is of the family $X = Ye^{-kt}$. (*Redrawn from Brody, S.* (1945).)

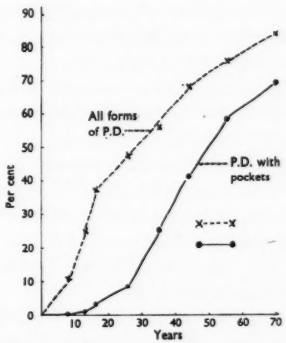


Fig. 9.—Age distribution of periodontal disease amongst 28,926 Americans (whites). x - - - - x Per cent positive—all unhealthy. ●——● Per cent with pockets. (*From Russell, A. L.* (1957).)

way, the annual incidence curve of periodontal disease *without pocket formation* seems to belong to Group A, whilst that for cases with pocket formation fits best into the class of Group B.

In selecting material to illustrate the age incidence of periodontal disease (*Fig.* 9) I have

studied a number of papers, and from amongst these have chosen that of Russell (1957) as being the most valuable for the following reasons:—

1. Russell's survey of a large sample of American population was a clinical study.

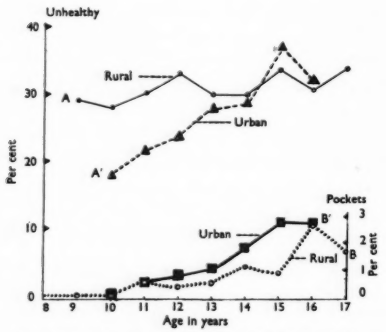


Fig. 10.—Age distribution of periodontal disease in children. *A* and *A'*, with unhealthy gums and teeth; *B* and *B'* with pocket formation also. (*From Benjamin, E. M., Russell, A. L., and Smiley, R. D.* (1957).)

2. A large number of persons (28,926 whites and 1018 negroes) were examined by a simple and standardized method.

3. These large series covered a wide age-span, and included approximately equal numbers of men and women.

4. The survey included a large proportion of adolescents up to the age of 19 years—hence the data for this part of the age-distribution curve should be reliable.

From his analyses, Russell rightly draws three main conclusions:—

1. That periodontal disease may appear in young children under 10 years of age, but is then not usually associated with pocket formation—presumably it is mainly gingivitis. (This is confirmed by other workers, e.g., the child survey of Benjamin, Russell, and Smiley (1957).) (*Fig.* 10.)

2. Both prevalence and severity of periodontal disease increase with age, in both sexes alike.

3. The disease is slightly more prevalent and progressive amongst men, with the possible exception of some part of the second decade.

It seems to me also that a proportion of the persons recorded by Russell as edentulous (hence not scored in his survey) must have lost their teeth from periodontal disease. Hence, in my further treatment of Russell's figures, I have made an allowance for this fact, correcting each age-group by a factor derived from the diagram given by Brekhus (1929).

In considering Russell's tabulated data, I have been mainly concerned with the rates of annual increase of periodontal disease in the sample of population that were examined. These people may be classified into four groups: (1) With normal healthy teeth and gums; (2) With unhealthy teeth, etc., but no pockets; (3) Those cases with pockets; (4) The edentulous.

It is here necessary to explain very briefly the statistical method applied to Russell's tables. In addition to what I have already commented about the edentulous, I have made

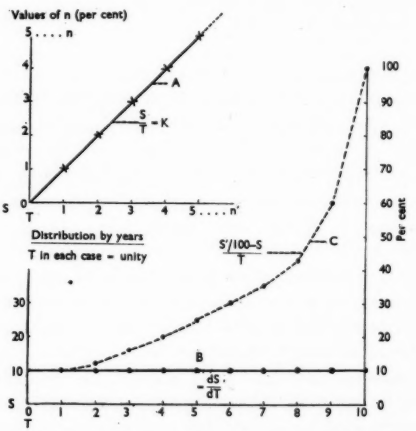


Fig. 11.—The relationship between rate of annual increase and annual distribution. A, Values of *S* at time *T*; B, Growth rate per annum; C, Specific growth rate per annum.

one further simple assumption, i.e., that periodontal disease will be progressive unless competently treated. This would seem to be reasonable, in view of the finding that the severity of the disease has been shown to increase with the years.

Fig. 11 A plots the relationship of two variables S and T on an arithmetic grid, where S varies as T for all values zero to n, the data for T representing an age/time scale. If we then replot the annual increase of S (as a percentage) we obtain curve B which shows a constant number of new examples of S occurring each year.

However, as mentioned elsewhere, one is primarily interested in the rate of increase of periodontal disease each year amongst the selected age-groups of a cross-sectional survey, and one may assume that the disease, once established, persists unless energetically treated. Hence one is primarily concerned with what occurs at yearly intervals amongst the residual healthy members of each age-group.

If one then regards S as representing some hypothetical disease as afflicting 100 young persons, the actual annual incidence figures of new cases would be as follows:—

Year	*Number "at risk"*	*Number with hypothetical condition S*	*New cases per cent per annum (= annual incidence)*
1	100	10	10 per cent
2	90	10	11·1 per cent
10	10	10	100 per cent
n	$100-S$	S'	$\frac{S'/100-S}{T}$ per cent

These data are plotted in *Fig.* 11 C, and show a steadily increasing incidence amongst the residual unaffected population.

In this present instance, however, we are also trying to obtain longitudinal significance from cross-sectional observations, hence I think one must consider what happens to the residual healthy members of any given age-group in the light of subsequent records, comparing all upon a per cent basis. Thus we see from *Fig.* 11 C that the per cent rate of increase of the lesion annually is not static as age advances, but actually increases with the passing years. (This is, of course, in my hypothetical case in *Fig.* 11.) *Fig.* 9 shows the crude age distribution of periodontal disease amongst whites, as found by Russell in his survey.

Fig. 12 shows the annual rate of increase of periodontal disease derived from the last plot, by the method of treatment which I have described. By examining these more closely we may perhaps get some idea of the connexion of the aetiological factors in various broad age-groups. This last diagram (*Fig.* 12) shows two curves, one for periodontal disease, all cases,

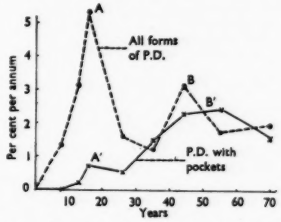


Fig. 12.—Rate of increase in incidence of periodontal disease per annum (whites). (*From Russell, A. L.* (1957).)

i.e., those with unhealthy mouths, and one for periodontal disease with pocketing. These curves depict the specific rates of increase of these two conditions for persons up to the age of 70 years (*from* Russell's Table 1).

It is interesting to note the bi-modal form of both of these curves, and the following tentative conclusions have been drawn:—

1. The annual incidence of periodontal disease in juveniles is not markedly associated with pocket formation. The milder gingival affection I would suggest is unlikely to be associated with loss of alveolar bone, and I have already expressed the opinion that it is probably mainly related to some external agency, conditioned by a hormonal factor, but not markedly influenced by systemic disease elsewhere in the general run of cases.

2. The smaller but coincident peak *A'* suggests that a few of these juvenile cases of gingivitis progress on to periodontitis simplex, with more severe lesions. Nevertheless, the great majority of these young persons probably recover, since it is not until a decade later that the rate of pocket formation curve begins to mount towards the high levels seen in older people. No doubt the majority of cases of severe periodontal disease seen amongst juveniles come from amongst those afflicted with the lesser complaint. There are also, however, a few interesting cases—such as those reported by Spirgi and Calandriello

(1959), which are atypical since the periodontal lesion presents as a degeneration rather than as an inflammatory condition. The pathogenesis of this small group is obscure, but according to these writers there was nothing to suggest a hereditary trait, or a disturbance of the calcium–phosphorus metabolism in their cases. In one patient, a boy aged 10 years, there was some biochemical evidence suggesting an abnormality of protein metabolism, but only in this boy. Quite clearly, these less common cases of juvenile periodontosis will require much further investigation before one is able to comprehend their causation.

3. The two curves of annual rate of increase in incidence of periodontal disease with and without pockets practically meet at about 35 years of age; after which there is probably no significant difference in their values. Hence, most fresh cases of periodontal disease when first seen will presumably have pockets already developed. This is a very different picture from that of children and adolescents, and suggests at once that there is some factor which operates after 35 years of age, so that the gingival lesion now usually progresses to the more severe state of pocketing. The only alternative explanation would be that a proportion of these older folk develop pockets *de novo*, without any previous gingival lesion. This, I believe, is actually so, but I have no idea as to the relative numbers of older persons who appear with periodontitis simplex and periodontitis complex, although I understand that the latter condition is probably more often seen in older persons than amongst the young. Nevertheless, from my brief study of the literature, I get the impression that the simplex form of periodontal disease is commoner, even amongst older patients.

The later shape of the two rate-of-incidence curves argues strongly in favour of a causative linkage again, maximal in the middle 30's or so; but with unusual recovery from the gingival lesion. This conclusion is, moreover, supported by Russell's clinical finding of increasing severity of periodontal disease with advancing age. As has already been postulated, it seems likely that this progressive nature of periodontal disease in older persons must be a reflection of their relative inability to repair and heal minor degrees of gingival damage, which in the young and more vigorous would resolve without further complications. The diminishing incidence rate of periodontal disease in persons over 55 years of age (if confirmed) does not suggest that tissue degeneration (e.g., osteoporosis) or systemic disease makes any great contribution towards the causation of periodontal disease, although doubtless they must play some part, most probably in contributing to the initiation of the cases of periodontosis.

Certainly, at this end of the age scale the percentage of persons afflicted with periodontal disease is maximal, but the annual morbidity rate per annum tends to drop—both in whites and negroes, males and females alike—according to Russell's survey. If senility of tissues were in fact a vital factor, I should expect the annual morbidity rate to continue to advance with increasing age.

SEX

I will now deal summarily with the possible bearing of sex upon periodontal disease. This is a factor which has its place in the aetiology of most diseases. This is well exemplified by the generally greater male liability to cancer, including malignancy of the skeleton. In the case of the latter, the effect of sex, I think, is largely due to the greater growth, longer growing period, and finally larger size of the male (Price, 1955, 1958). (This applies to most mammalian species.) However, when studying the effects of sex upon any lesion, one should not make the mistake of thinking only in terms of sex hormones and their possible actions. Nowadays, one can discern two aspects of sex, the genetic and the hormonal, the former being related to the unpaired *X*-chromosome of male cell nuclei. It is presumably this genetic difference which governs the sex differentiation of embryonic life, although the fœtus is, of course, subject also to certain effects from the maternal sex-hormones (and in some species also to those of a concomitant twin of the opposite sex). It is known that the gestation period for girls is several days longer

than that for boys, and their bone growth is slightly advanced compared with that of the male, even at birth. In most mammalian species, including man, the female has a greater expectation of life than the male. As a corollary, this must mean a greater male liability to disease, hence to death. It is thought that this is in some way connected with the unpaired male *X*-chromosome, since there are hereditary diseases, e.g., hæmophilia, which appear only in males, although transmitted by the females, who are presumably protected by the *Y*-chromosome which is absent in the male.

In several published surveys, including that of Russell, it is stated that periodontal disease is slightly commoner and more severe in girls during the second decade than in boys. This small difference one may attribute to the dimorphism of dental and skeletal growth; but the general increase in periodontal disease seen in both sexes after puberty is probably mainly due to some external agent, or structural peculiarity, to which the gingivæ and teeth of the adolescent are especially vulnerable compared with young children. In older women there appears to be no overall predisposition to periodontal disease, even including the risks attendant upon pregnancy. The somewhat greater incidence of periodontal disease amongst men agrees with the findings for many other diseases, particularly in the middle and later decades, and argues strongly against the idea that sex may be a factor of any great importance in the causation of periodontal disease. In the individual patient, the course of the disease, when once started, may be modified by some functional aspect of sex, e.g., pregnancy, and this may be reflected as an unusually rapid advance of the local lesion; nevertheless, after the cessation of growth it seems that both sexes are equally vulnerable to this common complaint. An analysis of Russell's data (1957, Table 3) certainly suggests that after 35 years of age periodontal disease is on an average more advanced, but slightly less frequent amongst women than amongst men of like age and race, and that these small differences are maximal during the 5th decade.

CONCLUSIONS

In conclusion, what may perhaps be learned from all this welter of words?

1. One should approach the study of the aetiology of any disease with as broad a view as possible, with some knowledge of "the soil", i.e., the general background of biology, and especially with an appreciation of the processes of growth, maturation, and ageing of the organism.

2. Periodontal disease is a complicated affair to which there are many contributory aetiological factors, not all of which are as yet clearly discernible; nor is it possible at present to disentangle their causative roles; nor does it seem that these several factors are equally important at all periods of life.

3. There are distinct differences in the pictures of periodontal disease in juveniles, in whom gingivitis is often resolved without further trouble, as compared with older persons, in whom it is usually progressive.

4. In my comparison of periodontium with periosteum, I have tried to show how modification in structure in analogous tissues may be accompanied by a change in behaviour. One of the key features to the understanding of all periodontal lesions is the realization of the confined space occupied by the periodontium, any increase in the cellular elements or fluid component of which must be at the expense of the normal formed tissue elements, bone, cementum, or fibres.

5. It is suggested that in juveniles the main causative factor of gingivitis is an external agent, the effect of which may be hormonally conditioned. In older persons, the vitiated processes of healing concomitant with advancing age would tend to promote extension of a primarily gingival lesion, thus leading to pocket formation in the untreated focal condition.

Acknowledgements.—The author wishes to record with thanks his indebtedness to Dr. D. G. Lyon for the provision of histological specimens for study, and for advice in the selection of "key" papers in the relevant literature; also to Dr. J. H. Middlemiss for the loan of the radiograph reproduced in *Fig.* 5A

The reproductions and photomicrographs are the work of Mr. J. E. Hancock.

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Histologic Study of Heterogenous Bone Implants in Human Beings: Preliminary Report

Despeciated calf bone paste is cancellous bovine bone fragments which have been stored in 20 per cent bovine plasma for a minimum of 30 days at 40° F. and mixed with 10 per cent fibrinogen.

Anorganic bone chips are prepared by removing the organic matter from the bone with ethylenediamine.

The reactions of the body to these substances were studied by inserting them into tooth sockets and subsequently biopsying the area after varying intervals of time.

Both types of material appeared to delay bony healing. Despeciated calf bone paste fragments were resorbed and the anorganic bone was here and there invaded by granulations, where there were wide Haversian canals. Eventually, it, too, was resorbed. Deposition of bone did not take place on the particles unless they were incorporated in bone growing in from the socket walls.—*J. oral Surg.* (1959), **17**, 3.

Penicillinase Treatment of Allergic Penicillin Reactions

The discovery of penicillinase in 1940 by Abraham and Chain is described and previous papers on treatment by this method are reviewed. Penicillinase injectable is supplied as 800,000 units of purified injectable hyophilized powder in single dose vials. It may be given intravenously for anaphylactic reactions, followed by a further dose by deep intra-muscular injection. Chills and fever have occasionally followed its use and redness and swelling at the site of injection, but no more serious side effects. It should be given within 24–48 hours of the reaction starting and within one hour all circulating penicillin will be inactivated. The effect is maintained for four to seven days. An illustrative case is quoted.—SPIEGEL, L. H. (1959), *J. oral Surg.*, **17**, 27.

NEW MATERIALS AND APPLIANCES

THE new materials and appliances briefly reported here have recently become available to the profession. For further information the reader should write direct to the manufacturer.

A Dental Bib Apron for Patients

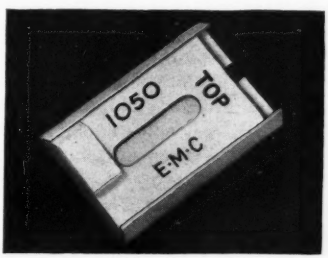
MADE in 45 denier nylon coated with polyurethane. Colour white. Weight 2½ oz. Will not crack or become sticky under any conditions. Tailored to fit over the shoulders with a three-button neck fastening and having the appearance of oiled silk. Easily cleaned with soap and warm water. Price: 22s. 6d. (Messrs. Cottrell & Co. Ltd., 15/17 Charlotte Street, London W.1.)

High Speed Burs

HI-DI high speed burs are designed for friction grip handpieces. There is a range of 18 burs of different sizes and shapes. They can be supplied in standard sets of six or singly. (Diamond Precision Tools Ltd., 105 Bolsover Street, London W.1.)

Radio-active Monitoring Service

A RADIO-ACTIVE monitoring service is now available for individuals who may be exposed to radiation in the course of their work. A



film holder with film is supplied, the film is returned at the end of each week for processing and assessment of dosage. If this should exceed the recommended dosage, warning is given so that the affected person may be treated and removed from the danger of further contamination. (Radiation Monitoring Service, Electronic Machine Co. Ltd., 41 Lodge Road, West Croydon, Surrey.)

The Ansafone

THE Ansafone is a simple instrument which answers an unattended telephone. On an incoming call the bell normally rings for 10–15 seconds, at the end of which the instrument

answers with a pre-recorded announcement identifying the subscriber and inviting the caller to leave a message which may be up to three minutes of recording time. If the caller pauses for more than ten seconds, the Ansafone informs him that "the machine is closing down" and automatically disconnects itself from the line. (Tele-Nova Ltd., 73 Gt. Peter Street, London S.W.1.)

Lignostab

LIGNOSTAB is a brand name for lignocaine hydrochloride. The cartridges are metal sealed, with a small hole through which the piercer needle of the syringe passes before penetrating the rubber diaphragm. Lignostab contains 2 per cent lignocaine hydrochloride. Lignostab A and N contain 1/80,000 adrenaline and 1/80,000 nor-adrenaline respectively. Price: Packets of 50 at 17s. 6d. or 200 at 62s. (Boots Pure Drug Company, Nottingham.)

MODERN MEDICINE

EXTRACTS OF PARTICULAR INTEREST TO THE DENTAL PRACTITIONER FROM THE MEDICAL PRESS

Reduction of Capillary Bleeding

Hæmorrhage can be a nuisance to the dental surgeon during oral surgical operations. This can now be easily reduced by the use of "Adrenoxyl" to produce capillary hæmostasis. It is indicated in surgical and medical conditions wherever capillary bleeding is encountered.

Adrenoxyl is a safe capillary hæmostatic which reduces the mean capillary bleeding time by decreasing the permeability and increasing the contractibility and resistance of the capillary wall. Adrenoxyl is the monosemicarbazone of adrenochrome in ampoule and tablet form. Being safe and giving rise to no side-effects, there are no contra-indications to its use.

Adrenoxyl does not affect blood-pressure or pulse-rate. It has no effect on the blood chemistry, and the clotting mechanism is unaltered. The action is confined to the capillary wall.

Dosage, 2–4 tablets to be taken the evening before operation, one or two ampoules of 2 mm. to be injected intramuscularly half to one hour preceding the operation. Two tablets can be taken three times a day post-operatively when the danger of capillary bleeding is present.

For children, one tablet is recommended to be taken the evening before operation, one ampoule of 2 ml. to be injected intramuscularly half to one hour preceding the operation. One tablet to be taken three times a day post operative when there is further danger of capillary bleeding. The above dosage may, of course, be repeated or increased if necessary.

Satisfactory results have been obtained in dental surgery by the use of tablets alone, although to obtain the optimum results both tablets and ampoules should be used.

Ampoules are supplied in boxes of six or of fifty ampoules for intramuscular or subcutaneous injection, each ampoule containing 0·75 milligram of adrenoxyl. Tablets are also supplied in tubes of 25, each tablet containing 2·5 milligrams of adrenoxyl.—RUDDELL, J. S. (1957), *Lancet*, **1**, 1092; ROLLASON, W. N. (1958), *Anæsthesia*, **13**, 56.

Air Travel

In these days, to save time when there is so much air travel to be done, it is important that everyone before flying has a check-up with his physician to find out if it is safe for him to travel by air. There are some diseases which preclude air travel, or necessitate precautions being taken during the journey. If the supply of oxygen to the lungs and the mechanical expansion of gases in the body is obstructed, air travel is not safe. A patient with artificial pneumothorax may fly if the mediastinum is fixed and a re-fill has not been given within 10 days.

Patients who have recently had abdominal surgery may be poor air passengers, as sudden decompression could cause severe complications.

If passengers with suspected coronary artery disease, anæmia, heart failure, or respiratory embarrassment propose flying, their physicians should carefully appraise them. They should also warn the aircraft company and carry a certificate of fitness. Slight deficiency of circulating oxygen at altitudes of 8000–14,000 ft. may cause hæmolysis in persons with sickle-cell anæmia.

Epileptics are more prone to seizures in aircraft when hypoxic. Diabetics must take a syringe, and insulin and glucose should be available in the passenger cabin.

Air-sickness is 5–10 times more frequent among children under five years of age than among adults, and is much more common among women than among men. Ear trouble is also most common among children because many young persons cannot be taught to make oropharyngeal adjustments during descent. Passengers must yawn or swallow to equalize pressure between their middle-ear and the atmosphere.

Equalization of pressure of the accessory nasal passages is basically automatic but may be obstructed by swollen or redundant mucosa or nasal polyps.

Persons with acute infections of the respiratory tract, allergic rhinitis, or nasal polyposis who must fly, should use a nasal vasoconstrictor or decongestant during flight, and particularly between ascent and descent.

Symptoms of air hunger are relieved by oxygen therapy. Dyspnœic type of respiration, which is frequently due to apprehension, and which is often misdiagnosed as air hunger, is alleviated by carbon dioxide inhalations or breath holding.—SPIEGEL, FREDERICK (1957), *J. Amer. med. Ass.*, **165**, 205.

Accidents

With the increasing death-rate on the roads it is important that drivers be warned about alcohol and about drugs, as free access to sedatives and hypnotics creates a source of danger to car drivers.

It is not realized that antihistamines and tranquillizing drugs can be very disturbing too. Antihistamines may cause drowsiness, and in the hay-fever season allergic patients who depend on antihistamines may be disturbed in the cerebral cortex.

It is recognized that some stimulants may help persons determined to travel great distances and who drive for very long periods, but their safety is very questionable. The emotional stability of the driver should also be considered.

The juvenile driver, however, under the forces of growth and broadening social horizon, demands, and usually obtains, free access to motor-car transportation. With his hurry, impatience, and enjoyment of the feeling of power and the freedom of movement, he can be a great nuisance to the public safety. Emotional stability, however, is not always obtained with passing time, and there are plenty of grown-ups who act with the same recklessness of youth.

The nervous tenison of a driver at any age is a factor which he alone must evaluate in determining the mode in which he operates his car and whether or not he can protect himself, as well as others, from possible accidents. —GRAEF, IRWING (1957), *Mod. Med. Minneap.*, **25**, 209.

Alcoholism

There appears to be no accepted definition of alcoholism but it can be defined as a progressive disorder of behaviour characterized by:—

1. Forms of drinking which deviate from traditional and customary use of alcoholic beverages in the community;

2. Loss of control over the use of alcoholic beverages; and

3. Interference with bodily and mental health, inter-personal relations, and social and economic functioning.

Alcoholics can be considered as sick people and yet there is no single disease pattern. Some reveal definite psychiatric disorders, in others it may be complicated by metabolic dysfunction affecting the liver, nervous system, and other organs.

To the alcoholic, alcohol comes more and more to occupy a central place in his life. It pulls him together in the morning, it calms him at night, it gives him courage, it provides escape, and he uses it to assuage the ravages of withdrawal.

Some people continue to drink all their lives with control and with no apparent harm, while others, sooner or later, are unable to regulate their drinking for which no single aetiologic factor has been found.

Unless the alcoholic controls his drinking he will follow a downhill course, terminating in death. It is, however, possible that a patient can become accessible to therapy at any phase of his illness. The most powerful motivating factor leading to effective treatment is that the patient himself realizes that he is unable to handle alcohol and that his life has become unmanageable.

Many alcoholics, however, can be helped to recognize their condition at earlier stages and thereby take effective steps towards complete recovery.—HOFF, EBBE CURTIS, and MCKEOWN, CHAS. E. (1958), *Med. Times*, **86**, 1.

BOOK REVIEWS

AN INTRODUCTION TO PERIODONTIA (The Postgraduate Dental Lecture Series). By HENRY M. GOLDMAN, D.M.D., F.A.C.D., SAUL SCHLUGER, D.D.S., D. WALTER COHEN, D.D.S., BERNARD CHAIKIN, D.M.D., F.A.C.D., and LEWIS FOX, D.D.S., F.A.C.D. $7\frac{1}{2} \times 4\frac{3}{4}$ in. Pp. 346, with 180 illustrations. 1959. St. Louis: The C. V. Mosby Co. (London: Henry Kimpton.) 58s.

DURING the past few years, Dr. Goldman has divided his original book on periodontology into two volumes, and it is not surprising that he has now found it necessary to produce what is virtually an up-to-date abridged version of the original, in which he has been assisted by those who contributed to his two volumes, with the addition of Dr. Chaikin.

Any book written by such authorities can be expected to contain information based on considerable clinical experience, backed by continuous research and analysis of the literature. This expectation is completely fulfilled in this book.

There are, of course, certain points of contention, such as the authors' extension of the term "periodontal pocket" to include any pathological change in the gingival attachment and also the use of the phrase "occlusal traumatism" to describe both the aetiological factor and the resultant pathological state. In both instances, careful explanation is given.

The habit of intelligent explanation which pervades this book considerably enhances its value, and anyone who reads it will be left in no doubt as to what facts are widely accepted and where it is necessary to make further research.

This book can be strongly recommended to anyone beginning to specialize in periodontology, and to those aspiring to a higher dental qualification, while the final year undergraduate student, who has received adequate basic instruction, will find it will help him to orientate his views. It is not, however, suitable as a basic text-book, as there is insufficient detail given about many of the fundamental procedures.

The standard of production maintains the high reputation of the House of Mosby, while limitation of the size, so that it will slip into a jacket pocket, is something which will please many people.

A. B. W.

NEW TEETH FOR OLD. By VICTOR H. SEARS, D.D.S., formerly Professor of Prosthetic Dentistry, New York University. $8 \times 5\frac{1}{2}$ in. Pp. 108. Illustrated. 1959. St. Louis: The C. V. Mosby Co. (London: Henry Kimpton.) 22s. 6d.

IT is not an easy matter for a professional person to write a book for the layman. If the terms used are too scientific it will baffle the patient and if the terms are too simple they will baffle the profession! Victor Sears has written this small book for the use of those many patients who find that they are to lose their teeth and wear artificial dentures. It is well known that the proper functioning of dentures requires the active co-operation of the patient, and it is essential that the correct advice is given before the treatment is commenced. The book deals with all the aspects of the reasons for extraction and leads up to the moment when the patient must eat his first meal using artificial means.

Nobody will agree with everything in this book and many will be amused at the illustrations, but there is little doubt that it will impart a great deal of useful information to the patient. It is written in an easy readable style, in the simplest of language, and can undoubtedly be recommended to all patients who have to undergo the penalty for gross dental disease. In a busy practice there is not always time to give a patient those detailed instructions to ease his mind, and yet if we do not give this advice the patient will be constantly on our doorstep.

The recommendation or presentation of this book to our patients will save us and our patients a good deal of time and worry.

N. L. W.

INHERENT ERRORS IN CEPHALOMETRIC FILMS AND THEIR REDUCTION

By **Professor G. E. M. HALLETT**

THE development of cephalometric analysis by means of X rays in the late twenties suddenly gave a new tool for the examination of the cranial skeleton. No longer was it necessary to compare an assortment of dried and anonymous bones whose different ages could only be determined within broad limits (the cause of death being quite unknown), in order to formulate a pattern of cranial growth. When measuring the same individual over a growth period, considerable lack of precision had to be accepted due to variations of individual assessment, the adjustment of instruments, and also to variations in mobility of the soft tissues overlying bone at different times of life. Photography could help with profiles and over-all outline, but here, too, inaccuracies arose all too easily, partly through lens aberration, and the fact that a single lens can only "see" tangentially so that particularly in stout people a quite false outline results. These deficiencies can, in part, be remedied by using high quality lenses and by photographing the patient from a considerable distance.

At first, the head-plate seemed to get over these troubles. The X rays penetrated bone and gave us suture points and bone borders, and, because they penetrated bone, the full limits were clearly seen. The old points of anthropology and biometry were quickly identified, or remodified together with a host of eponymous planes and angles, and other points. A great number of analyses depending upon geometrical constructions were invented to provide diagnoses from the relations of the bony structures to each other, and malocclusion became a phenomena almost entirely of skeletal structure and origin. To help to make these head-plates as accurate a record as possible Broadbent constructed his well-known cephalometer, which combines the accuracies of an expensive lathe and a high-class optical instrument. This instrument allowed fixation of the head by means of ear-rods, which were both alined on the central ray of the tube which was 60 in. away from the mid-sagittal plane of the patient. By exposing successive head-plates over a period of several years the films could be eventually superimposed and thus growth and tooth movements (artificial and natural) studied. Also, the soft tissues could be examined by suitable screening or other devices. However, to begin with, the soft tissues were rather ignored. Nevertheless, for the first time, a study could be made of the same individual from birth to maturity, and this was a most exciting advance. Since these early beginnings a vast amount of literature has been produced about roentgenographic cephalometry, as it came to be called, and those who have perused the weighty synoptic review of Krogman and Sassouni (1957) will realize the way in which the method has dominated orthodontic thinking and the valuable information that it has given us. But it has not all been advance. Many workers have become so engrossed with this tool that their minds and critical faculties tended to become first hypnotized and then paralysed by it. Diagnosis became a matter of planes and angles, the latter being sub-divided into seconds of arc and lengths measured by millimetres and parts of a millimetre. Arbitary points of superimposition were defined from which it was inferred that all growth takes place centrifugally, a very popular area being somewhere in the body of the sphenoid. Curiously, when the biometrist studies serial growth of the body as a whole he measures standing height from the floor; he does not usually put a pin through the umbilicus, though perhaps he should! However if we

Presidential Address given at the meeting held on Jan. 12, 1959.

are going to use the method at all we must have a "superimposition" point somewhere. The important thing is not to make it too sacrosanct. The other thing to remember is that what we are studying is a shadow picture or, to use Greek roots to say the same thing, a "skiagram". What we see are projections of our hard and soft tissues, and, because the latter have depth and breadth and because they must necessarily be a distance from the sensitized film varying from $1\frac{1}{2}$–6 in., every recorded shadow registered must be falsified to a greater or lesser degree. It is very like the camera problem previously mentioned. Our image is both enlarged and distorted. There are other troubles inherent in the cephalometric head-plate which I can only touch on here and some of them are again those of the photographic method.

1. The patient may move, producing a fuzzy image. The cure is stabilization of the patient (the cephalostat), arrest of breathing during exposure, and as short an exposure as possible. The latter implies a high-power machine.

2. The ray source may move, again blurring the image. The cure is obviously a well-constructed and solid mounting for the tube.

3. Short exposures which, as so often in photography are obviously desirable, are essential for cephalometry by X ray and are also helped by using fast films and intensifying screens. Fast films, however, tend to have coarse grain, and intensifying screens, by reacting to X rays and producing light, render the image less sharp because of halation.

4. Loss of definition is also caused by secondary radiation and this is reduced by using suitable hollow cones and cylinders to limit the ray bundle size leaving the tube and also by other devices at the film end such as the Schönander and Potter-Buckie screens. The Schönander screen, whilst improving the image, has the disadvantage that the grid shadow can be seen on the film. The Potter-Buckie gets over this by using a grid which moves during exposure. The disadvantage of the Potter-Buckie grid is that it interposes a further 2 in. between the patient and the film, thus increasing the enlargement error.

5. Loss of definition is also brought about by the penumbra due to the size of anode being bombarded. This error is reduced by having (*a*) the tube as far away from the patient as possible, and (*b*) by using a high energized tube with a rotating anode so as to give a focal spot of 1–$1\frac{1}{2}$ mm. square. At 60 in. and more, the penumbral problem is virtually eliminated.

6. We are left with the errors of magnification and distortion. Again these are reduced by having the greatest practicable distance between the tube and the patient (Korkhaus has suggested 13 feet) and the shortest between the patient and the film. Both are minimized by having the patient's sagittal plane at right angles to the central ray. The other errors are reduced by the best combination of kVA, stability, screening, and the head fixation possible.

A patient being X-rayed for a head plate should be comfortable and his radiation dosage should be the minimum possible. In general, I personally like a high kVA machine (80), 100 ma. 92 in. target/film distance, fast film, and a Potter-Buckie grid which allows an exposure of from 0·6 to 1·2 sec. A comfortable cephalostat is a great advantage and is a good thing if arrangement is made for keeping the mid-sagittal plane a constant distance from film and anode.

These errors have, of course, been known ever since X rays were used, but not many determined efforts have been made to reduce those of enlargement and distortion beyond stipulating a 60-in. distance or greater between cathode and film and very accurate positioning of the head in relation to the central ray.

Adams (1940) investigated the enlargements and variations of angles in head-plates by X-raying a series of skulls bearing metallic implants. The precise relations of these implants were measured on the skull by means of a special Western Reserve cephalostat. He also constructed a special corrective scale which would be mounted in the mid-sagittal plane and thus produce an image on

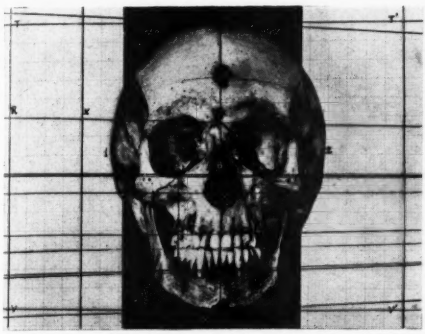


Fig. 1.—Showing natural skull mounted in relation to two film positions "X"=72 in. and "R"=74 in. Divergent black lines are taut threads stretched to a focal point actually at these distances so that the angles subtended are accurate. The displacement of the various points and their corresponding enlargement on the two film positions can plainly be seen by comparing with the parallels on the graph paper at T–T′ and V–V′. The three main planes for which correction was attempted are shown at A, B, and C. The range of magnification between points **1** and **2** is from 1·12 per cent to 9·6 per cent at X and from 4·2 per cent to 13·8 per cent at R.

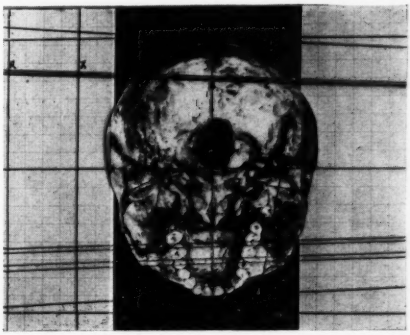


Fig. 2.—Shows the same skull in plan. Note the displacement of the right and left molar shadows and how their true mediodistal diameter will *not* be registered at all, only the greatest diameter from distolingual tangent to superimposition on premolar shadows. At A, B, and C, three hypothetical points at right angles to the sagittal plane and in the same line become three separate points on the film, either at X or R.

the film. Amongst his conclusions was the statement that corrective scales should always be used in cephalometric appraisals. Adams

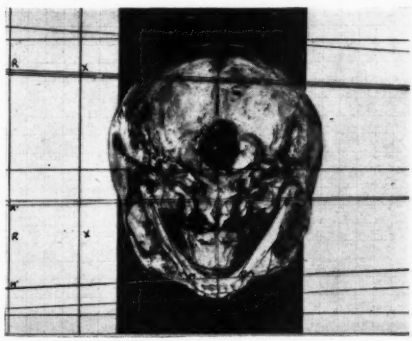


Fig. 3.—The mandible has been superimposed. Note how the obliquity of the mandible lengths A–M and B–O distorts the shadow at R and X reducing the magnification. In this case A′–M is equal to A–M whilst it is markedly less than B–O which is the side nearest to the tube.

examined 50 skulls by this method, and tabulated his results.

Thurow (1951), in an informative paper, discussed cephalometrics in private practice and many of the sources of error. He suggested using a specially adapted vernier on a slide rule to make the required corrections for distortion and magnification where special serial studies were being made to high degrees

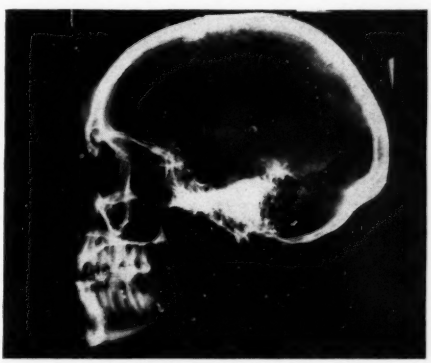


Fig. 4.—Skull with main points marked by small amalgam inserts. Measurements made on this specimen were unsatisfactory for the reasons given in the text.

of accuracy. For ordinary assessments he suggested correction to be unnecessary.

Franklin (1952), in an excellent contribution, discussed many of these factors again

and in particular illustrated the geometry of enlargement and penumbral shadows. Beyond the usual recommendations of a long tube/patient distance and a short patient/film distance he made no other comments on solving the problems of distortion and enlargement.

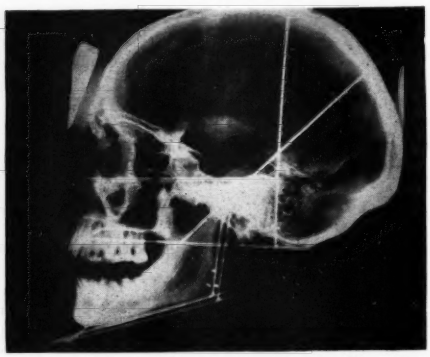


Fig. 5.—Skull with main planes indicated by calibrated rods. This gave more precise results regarding angles and linear enlargements, but the sagittal plane could not be accurately determined without sectioning the whole skull.

Kean (1958) examined a series of cases, Class II, division 2 malocclusion, for "aspects of facial depth". In order to make accurate measurements on his head-plate he felt that correction would have to be applied to his headplate films, and he constructed an ingenious millimetric scale which was radio-opaque and, placing this in the mid-sagittal position, he X-rayed this and used the superimposed scale to read off his dimensions in true millimetres.

To my mind this was the first practical attempt to apply corrections to the head-plate for enlargement error. Elaborate tables involve very tedious correlation and plotting which means that they can only be used for isolated measurements. The Wylie compensator has certain advantages but was designed

Table I.—Some Observations on Cephalometric Films exposed at 72-in. Anode—Buckie Distance on (1) Bony Skull (*see Fig.* 5); (2) Plastic Skull (*see Fig.* 6)

	Bony Skull			Plastic Skull		
	Actual	Film	Per cent mag.	Actual	Film	Per cent mag.
Skull height	135 mm.	146 mm.	8·14	112·0 mm.	120·0 mm.	7·1
Basic length	176 mm.	190 mm.	11·14	130 mm.	142·5 mm.	10·4
Right Frankfurt (horizontal)	90 mm.	91 mm.	1·1			
Left Frankfurt (horizontal)	100 mm.	104 mm.	2·5			
S.N. Bolton pt. angle				13°	13°	Nil
Mandible						
Right						
Ramus (horizontal)	112 mm.	112 mm.	0	78 mm.	85 mm.	8·97
Ramus (vertical)	55 mm.	57 mm.	3·63	54 mm.	56 mm.	3·7
Gonial angle	111·5°	113·5°	1·8	110°	111·5°	0·8
Left						
Ramus (horizontal)	112 mm.	112 mm.	0	78 mm.	85 mm.	8·97
Ramus (vertical)	55 mm.	59 mm.	7·26	58 mm.	65·5 mm.	7·7
Gonial angle	112·0°	114·0°	1·8	110°	110·5°	0·454

mainly for A.P. films. For some years I have been particularly interested in projection error (*Figs.* 1–3) and having over the last twelve years accumulated a considerable number of serial lateral skull films, have been interested in examining them for growth and for changes following orthodontic treatment.

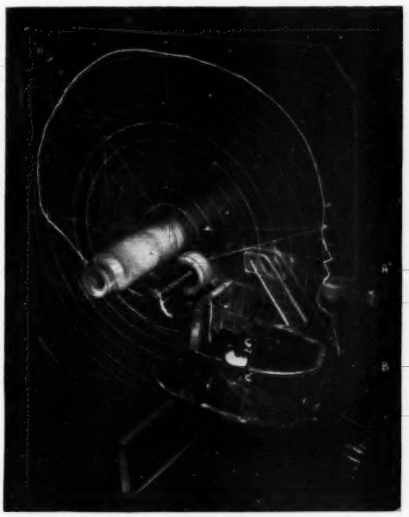


Fig. 6.—Perspex skull. The sagittal plane could be isolated for copying, and could be scribed with radio-opaque points and lines. The mandible and other points away from the median plane could be exactly symmetrical and variable within precisely determined limits.

I was not able, however, to embark upon the vast quantity of correction that tables and individual plotting would require and therefore made some investigations of my own. I started, like Adams, with a natural skull, using metallic implants (*Fig.* 4). This method, however, was rejected. The implants had to be too thick and clumsy and changes in orientation of a cylinder, however small, altered its image and dimensions, thus making for lack of accuracy in plotting points. In order to improve on this, a natural skull was taken and the main planes were put in in the form of fine metal rods (*Fig.* 5) (fine orthodontic tubing). Separate rods were used for the right and left Frankfurt horizontals for the right and left mandibular planes, posterior planes, for length of cranial base, height of skull (basion to bregma), etc. These wires were divided into centimetres, some by having fine cross cuts at centimetre intervals, and others having very fine stainless steel wire welded on to them. Measurements made from this skull are shown in the three columns of the left side of *Table I*. The following observations were made from the films of this skull:

Sagittal dimensions crossing central X-ray axis increase from 7–8·5 per cent.

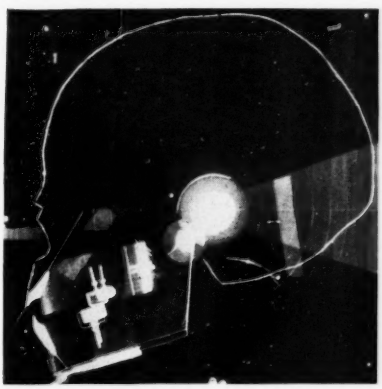


Fig. 7.—Head film made from perspex skull mounted in the cephalometer and radiographed.

Sagittal dimensions away from central X-ray axis increase from 10–11 per cent.

Sagittal angles alter too little to make any significant difference. Lateral angles, e.g., gonial, away from central axis open 1–2° (agree with Adams). This may open mandibular/Frankfurt or mandibular/palatal plane angles. Divergence of lines from central point tends to cancel out enlargement by distortion (*see Fig.* 3). This is a very variable factor differing between individuals and depends on the degree of obliquity of horizontal rami from the medium plane. After this, it was decided to construct a synthetic and symmetrical skull from perspex (*Fig.* 6). This could have lines, angles, and concentric circles marked upon a sagittal plane (unattainable with the natural skull). The lines were scribed and then filled in with white lead

paint (heavy-bodied) to make them radio-opaque. Certain parts were made movable using screws so that the movements could be precisely determined. For example, rotation of the "molars" C through $2\frac{1}{2}$ turns was equal to an "eruption" of 1 mm. The skull was mounted in the cephalostat and exposed in the normal way. The resultant film could then be examined without having a great deal of obscuring detail. The procedure was as follows: A head-plate film was numbered and the film exposed (*Fig.* 7). Various adjustments were then made either by moving the molars through a definite distance upwards or downward or by dropping the mandible a precise distance by turning the nut A on the threaded and spring-loaded rod B through a definite number of revolutions. Further films were numbered and compared. These films could be superimposed exactly upon each other, and then by dismantling the skull and laying the sagittal portion on a film and exposing again a "contact" print could be made. The enlargement errors could then be precisely examined between actual size and film size and also the error between the actual movements and the recorded movements. *Fig.* 8 shows two superimposed tracings. The inner one is made directly from the sagittal plane of the perspex skull, the outer one is the tracing of the head-plate made as shown in *Fig.* 7. From this figure we can see plainly: (*a*) the overall degree of enlargement; (*b*) the successive and increased displacement of points from the centre to the periphery (line P–O).

The right angles scribed in the occipital quadrant show no detectable change of angle between actual and film registration (they are of course displaced centrifugally). Another experiment was to expose two films without altering the "skull" at all. The first was made with the central ray alined on porion (*Fig.* 7), the second with the central ray shifted to the canine region. Studying the results of measurements made on successive films from the plastic skull leads to the following conclusions: Skull measurements of 1–$1\frac{1}{2}$ mm. are not clearly differentiable on the film to decimals of a millimetre. This means that, particularly in the molar region, it is invalid to try and work to less than a millimetre of accuracy.

Change of central ray by $2\frac{1}{2}$ in. produces profound film change registration of central points, and points up to molar distances from porion and also in the relative film registrations of near symmetrical points (R. and L. molars) by parallax. (Note *Figs.* 7 and 9.)

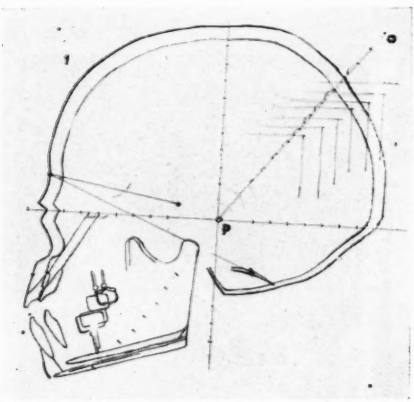


Fig. 8.—Tracings made from former film (outer line) and actual median perspex plane (inner line).

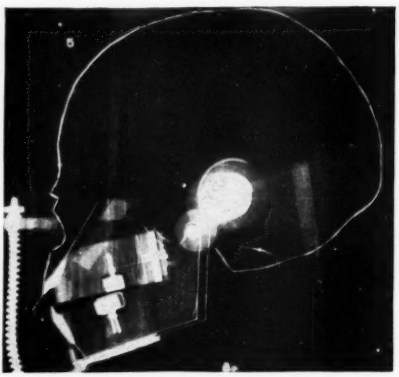


Fig. 9.—Head film made after tube had been moved horizontally $2\frac{1}{2}$ in. towards the "canine" region. Compare with *Fig.* 7 and note changes in "molar" shadows and widening between posterior planes of mandible.

Change of central ray by this amount produces remarkably little change in the recorded profiles, i.e., between the profiles of *Figs.* 7 and 9.

Magnification follows clearly-defined mathematical laws, and the extent is given by this formula:—

$$\text{mag.} = \frac{D}{D-d} - 1 \times 100$$

where D = distance from anode to film
d = distance from object to film.

It follows that there will be considerable differences in enlargement between the right side of the skull and the left. For example, if the film is placed at 72 in., i.e., as close to the head as possible without interposing screens, the enlargement will be as follows:—

Film side molar	*Median points*	*Tube side molar*
3·5 per cent	5·1 per cent	6·66 per cent

With the Potter-Buckie interposed, percentage magnification is correspondingly increased as follows:—

Film side molar	*Median points*	*Tube side molar*
6·47 per cent	8·0 per cent	9·63 per cent

A considerable number of other measurements were made, which it is not proposed to discuss here, except to say that it became apparent that the interpretation of fixed lines and points under almost ideal experimental conditions still resulted in inaccuracies, even when plotted with the utmost care using high-grade instruments. It was concluded that when working upon a living person where conditions are complicated by (*a*) growth, (*b*) slight variations in head position due to separate fixations in head positions at varying time intervals, and (*c*) slight variation in porion alinement due to soft tissue mobility around ear-plugs, a considerable amount of inaccuracy must be accepted and only very definite and marked variations from previous records accepted as significant.

The next stage was to find whether an instrument could be constructed to "recover" from the film a plotting of the essential features in such a way that would be obtained if all the X rays were parallel and not divergent, i.e., to cancel out, or markedly reduce, the magnification factor from an average of 6–10 per cent to about 1 per cent. It was felt that if this could be done easily and automatically, the results of tracings would have greater value where accurate deductions had to be made. A pantograph seemed to be the obvious instrument, but no suitable one was available commercially and a series of pantographs were constructed with successively different

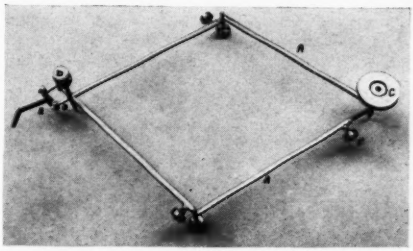


Fig. 10.—Pantograph for correcting enlargement error.

modifications until one was found to give the required results. This not only had to satisfy the theoretical mathematics of the problem, but also had experimentally actually to "recover" from a film of the plastic skull, exposed at 74 in. anode film distance, the exact sagittal points scribed upon the central perspex plane. It was found that the pantograph illustrated in *Fig.* 10 would do this. The next stage was to be able to correct for magnification in two other main planes, i.e., right and left molars (A and C, *Fig.* 1). At first it was thought that three separate pantographs would be necessary, but it was found that an adjustable one could be constructed to satisfy the requirements.

The main framework of the instrument is constructed from square German silver tubing, as used for maxillo-facial work, with round tubing for the pivots and hinges, the whole assembly being hard soldered. By using square rods to fit the tubing for the major sides an extensible large square results, extension being controlled by circular screw stops on each side. The pin on the major square is sharpened so as to pierce through porion on the film and is weighted to hold it easily in place (*Fig.* 10, C). A 6H "Eagle" lead, suitably sharpened, fits into the tubing joining the

minor and major square, and thus forms a pivot between them. The lead carries a brass button (D) which can be pressed on lightly with the finger during use, thus compensating for wear. The tracing needle (E) on the minor

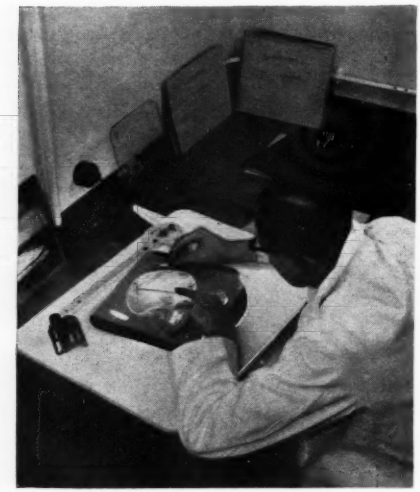


Fig. 11.—The pantograph in use on the rotating viewer. (The instrument shown is one of the earlier models made of round tubing.)

square (B) (0·36 in. side) is made to follow the outline on the transilluminated film, and the pencil then traces the true outline which is variably reduced according to the distance from the centre. The larger the major square the less the amount of correction. Three marks are made on the square rod, thus giving three separate corrections:—

1. The largest square is used for the film side of mandible, and for all cheek teeth (5·5 in. side).

2. The intermediate square is used for all sagittal points (4·75 in. side).

3. The fully closed square for mandible and cheek teeth (posterior to canine) on the tube side (4 in. side, A).

In order to use this instrument easily and efficiently a special viewer was constructed (*Fig.* 11). This employed a 12 in. circular fluorescent light behind a circular plate of glass mounted horizontally. The circular plate revolves on circumferential rollers and is flush with the surface of the tracing table surrounding it.

A small central hole is drilled through the glass plate and three other small holes at about 3–4 in. from it. A head film with an overlying sheet of tracing paper is then pinned to this plate by piercing ordinary pins through tracing paper, film, and glass. The central pin is then withdrawn, the weighted pin of the pantograph inserted in its place, and the

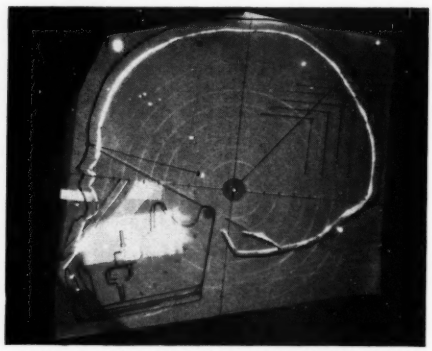


Fig. 12.—Superimposition of: (1) Tracing of head-plate 1 (solid outer line); (2) Actual perspex median plane with scribed lines (white); (3) Tracing by pantograph from outer line (faint black line superimposed on white line).

tracing now made. The head-plate and its supporting circular glass plate are rotated as convenient for right- or left-hand tracing. By suitable masking the soft tissues can be easily distinguished and it is a help to mark these on the actual film with an HB lead before reducing with the pantograph.

The first requirement of the pantograph was to take the actual sagittal perspex plane and a tracing of the head-plate film of the perspex skull so as to give *Fig.* 8, as previously explained. This was now centred through porion on a film of the first exposure (*Fig.* 7) and with superimposed tracing paper mounted on the rotary viewer as in *Fig.* 11. The profile and certain sagittal points were followed with the tracing needle of the pantograph and the resultant pencil trace should then follow the accurate inner correct line. This is not easy

to show in illustration, but *Fig.* 12 was the result after successive empirical modifications of the pantograph. The pencil line overlying the actual white lead scribed on the sagittal perspex can just be distinguished.

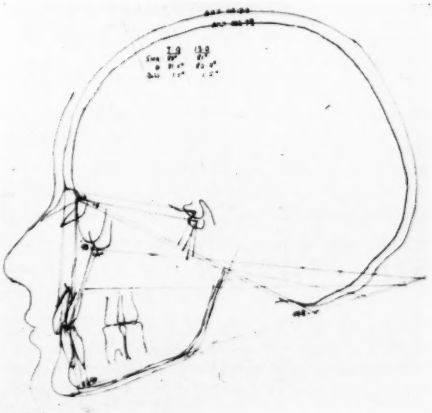


Fig. 13.—Pantographically reduced tracing of two serial head films for girl at ages 7 and 13 years.

Having attained this degree of accuracy, actual head-plates from live patients were traced.

Fig. 13 shows the corrected serial tracings of a girl at 7 years 9 months and 13 years of age and *Fig.* 14 shows these tracings superimposed upon the original 13-year head film, showing the amount of compensatory correction.

Fig. 15 shows another girl between the ages of 6 years 3 months and 13 years 2 months: The solid lines are the corrected tracings and the outer dotted lines the actual tracings from the head-plates. From these two cases showing different vectors of facial development, interesting measurements can be made on the corrected tracings which will have greater validity than these made directly on the original films.

It is not suggested that all head films should be corrected in this way, but where accurate measurements have to be made to find out what has been accomplished by orthodontic movement or by growth changes it must be important to have the minimum of magnification and distortion error. It is felt that the instrument described makes a contribution towards this, and further work will be done to make it as accurate as possible. It should be borne in mind that rarely will the same instrument supply the correct compensations for another cephalometric set-up. Tests have to be made as described, and the necessary

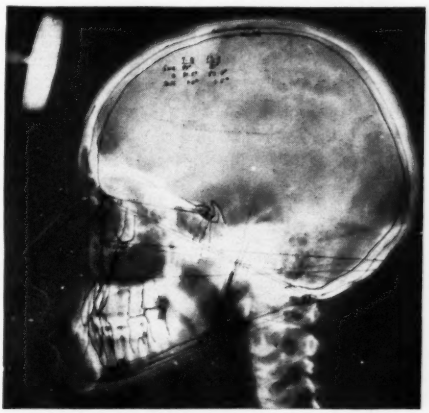


Fig. 14.—*Fig.* 13 superimposed upon actual age 13 years head film.

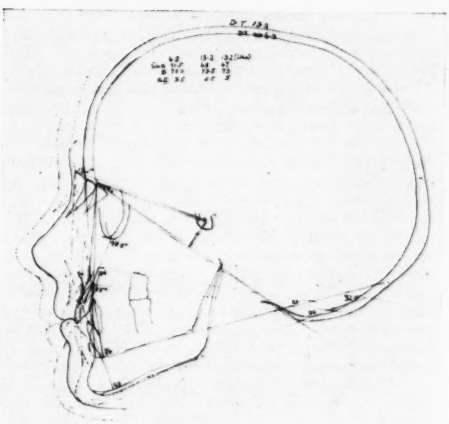


Fig. 15.—Tracings of serial head film for girl at ages 6 years 3 months and 13 years 2 months (solid lines—pantograph: broken lines—direct tracing from age 13 years 2 months head film.

adjustments made. However, by making the major arms adjustable, this should not be difficult in the first place.

Acknowledgements.—I am indebted to Mr. S. V. Haswell, Consultant Radiologist to the Newcastle Dental Hospital, for the facilities of his department, and also to Mr. Watson, Radiographer, for his help and co-operation. I would also like to acknowledge the kind help I have had from Commander (E) D. G. Thompson (Retd.), who made valuable observations of my original pantograph, suggested its present basic form, and helped me with some of the calculations.

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LETTER TO THE EDITOR

Sir,

May I suggest, with reference to your stimulating editorial on the toothbrush and dental health (May, 1959) that you omitted to stress an important aspect, viz., the *manner* in which the brush is applied. This has a direct bearing on its constitution and shape, and is the main stumbling-block to uniformity of approach to the problem.

The subject has been seriously examined for decades and a glance at the background helps to define the basis of the present diverse opinions; also the unlikelihood of getting an impartial view which will reconcile them. From North America the names of earlier oral hygiene enthusiasts like Charters, Stillman, and Box, who had a profound influence, spring at once to mind. Although there have always been differing ideas on methods, the aim of each was the same: the application of bristles to the clinical cervical regions in such a way that damage, especially to the gum margins, was avoided. This was accomplished in one of two ways: (1) either by applying the sides of fairly resilient bristles with the brush head moved in various directions, or (2) by accepting the almost universal scrubbing action and supplying a soft brush. The first allowed for very little latitude in their exact performance and were probably more suited to the unusually conscientious individual: the average person does not display a marked ability to acquire new habits involving manual dexterity. The much-propagated rolling technique, whereby the sides of the bristles are applied above the gum margins then swept toothwards by turning the wrist, has long been the most popular in this category. The second way is exemplified in the realistic method of oral hygiene advocated by our own periodontal genius, Sir Wilfred Fish, some twenty-five years ago. The difference between these two concepts gives rise to much of the uncertainty about which lead to follow and what type of brush to use—whether to be a "sweeper" or a "scrubber", which is evidenced by the periodic pleas for agreement. The provision of any designated brush on the other hand, thanks to the willing co-operation of manufacturers, should be relatively simple. Incidentally, the object produced by patients for inspection is often merely a dirty, shapeless mass of splayed-out nylon filaments.

May I further suggest that the least complicated approach to mouth hygiene is aided by the oldest firm of brush manufacturers who produce a specially shaped toothbrush made of soft horsehair which retains a functional shape for months. An adjustment of the natural action is fairly easily taught and subsequently supervised. They are cheap enough to be kept in bulk and supplied at the time. The wrapping around each brush is illustrated herewith:—

A, This brush is trimmed to a wedge shape to enable it to reach easily into crevices. B, The brush should be used with a light scrubbing action, when the long central bristles reach into crevices with the minimum irritation.

To anyone interested I will gladly send samples of this brush.

Yours sincerely,

K. MCALLISTER.

4, *Saxby Street*,
Leicester.

ABSTRACTS FROM OTHER JOURNALS

The Lower Incisor—Its Influence on Treatment and Æsthetics

A comparative evaluation is made of various methods of relating the lower incisor to the skeletal and soft-tissue profile. Tweed suggests that the lower incisor should be at approximately 90° to the mandibular plane when the Frankfort–mandibular angle is favourable, or that the lower incisor should meet the Frankfort plane at about 65°. Steiner measures the distance of the most labial portion of the lower incisor crown from the line NB. He also measures the angle which the lower incisor makes to the NB line, the average for which is 25°. Downs has a method of measuring from the incisal edge of the lower incisor to the line A–Po; that is the lower incisor is indirectly related to the chin point. In Holdaway's method the lower incisor and the chin point are both related to the line NB (extended). Ideally, the two measurements should be nearly equal. Eight cases showing dramatic tooth movement are analysed according to the four methods, but the method of superimposing the X-ray tracings should be noted. The before and after tracings were superimposed on SN at N and pre-treatment lines and planes transferred to the second tracing. The second tracing was moved upwards until the anterior nasal spines were level. In addition, composites of the maxilla were superimposed on the incisal floor and the mandibles on the profile image of the symphysis and the lower border.—LINDQUIST, JOHN T. (1958), *Amer. J. Orthodont.*, **44**, 112.

Bone Graft in the Treatment of Intrabony Periodontal Pocket in Dogs—A Histological Investigation

Intrabony pockets were created in 10 dogs of both sexes, of unknown age and parentage. The method of creating these pockets was to reflect buccal and palatal flaps, and then with a fissure bur to cut a V-shaped intrabony defect along the mesial surface of the tooth to a depth of from 3 to 4 mm. and about 2 mm. wide. The space created was filled with self-curing acrylic, tin foil, or surgical cement, before the flaps were replaced and sutured. The acrylic material produced considerable amounts of inflammation and bone loss and was, therefore, discontinued.

Two pockets were created in the same jaw of each animal, one on the right for the bone-graft experiment, and one on the left for the control.

The bone-graft operation was performed one week after creation of the pocket, fresh autogenous cancellous bone being used. This bone was obtained from a site between the maxillary precanine and the canine.

It was found that bone regeneration and connective tissue re-attachment occurred. Osteogenesis took place on both the graft and the parent bone. The amount of regeneration and re-attachment appeared to be in reverse proportion to the severity and duration of inflammation. It is believed that bone grafts may be successful even when the bifurcation is involved.

Even in the absence of a graft healing occurred, but only a very small amount of new bone was formed, although the longest period of observation was only 16 weeks.—YUKTANANDANA, I. (1959), *J. Periodont.*, **30**, 17.

The First Permanent Molar Again

This author is as equally enthusiastic today about the timely removal of the four first permanent molars as he was eighteen years ago when his article, with the same title, appeared in the *British Dental Journal*. He advocates the treatment for every case save one, and that is one where the dentition is already incomplete as shown by radiographs. Only once has he seen a mouth with 32 teeth which was not over-crowded.

The treatment starts at the age of $8\frac{1}{2}$ years when the jaws are X-rayed; at this age the crypts of the third molar teeth usually can be spotted. If a full complement of teeth is

found to be present, extraction of the four first permanent molars is decided upon and not delayed beyond the age of 9½–10 years, and the earlier the better. At this early age the second permanent molars are only half-formed and when the tooth is in this state (H. G. Watkin is quoted here) it moves vertically in the alveolus and does not tip.

Results of the treatment are: (1) Caries reduced by more than 80 per cent; (2) Impacted 3rd molars are unheard of; (3) Absence of any disfiguring irregularity in 95 per cent of cases; (4) Greatly reduced tendency to periodontal disease.—WILKINSON, A. A. (1959), *S. Afr. Dent. Ass.*, **14**, 179.

Fibrinolysis

Fibrinolysis is an extremely rare blood-coagulation disorder which consists in the dissolution of the clot of fibrin formed when blood coagulates. If blood is collected from a patient following a surgical operation, following a hæmorrhage or an adrenaline injection, or from a patient who has secreted adrenaline through fear, and if the blood is then allowed to clot and stand at 37° C. the fibrin is dissolved.

In the normal process of hæmostasis the clot is, of course, only a temporary framework for the growth of fibroblasts and new capillaries, and as new tissue forms the fibrin disappears. The body thus possesses a means of disposing of unwanted fibrin. There is evidence that this is achieved by powerful precursors in the blood itself. Fibrinolytic activity may be induced in the body by certain bacterial filtrates, chloroform, post-mortem blood, and by physical stress. It is not known if the fibrinolysis is due to the same lytic factor on each occasion.

A case is described of an intractable hæmorrhage following a root extraction and which, in the light of the case history, was considered to be due to hypertension and which continued because of fibrinolysis. At no stage during this hæmorrhage did the blood issue from a particular point.

In commenting on the condition the author says that MacFarlane observed increased fibrinolytic activity in human patients after surgical operations and, later with Biggs, found that this activity was more related to the mental distress of the patient than to surgical trauma. It has been observed also in other distressing circumstances, such as air raids and anxiety states and under hypnosis.—MACANSH, J. D. (1959), *Aust. dent. J.*, **4**, 90.

Tarnish of Dental Alloys

In an investigation conducted to determine the roles of chemical elements in the tarnishing of certain dental alloys, the following facts were established: (1) Sulphides were shown by X-ray diffraction patterns to be a dominating factor; (2) Amalgam was grossly discoloured in sulphide solutions, but to a lesser extent in chlorides, synthetic saliva, and water; (3) The degree and rate of tarnish did not appear to be influenced by residial mercury or zinc content; (4) Gallium alloy was little affected by sulphur, but badly by chloride, hydrogen peroxide, and distilled water; (5) Sulphides cause a light tarnish on low carat gold but high carat gold was unaffected.—SWARTZ, M. L., PHILLIPS, R. W., and TANNIR, M. D. (1958), *J. dent. Res.*, **37**, 837.

SEMAINE ODONTOLOGIQUE INTERNATIONALE

The 64th Dental Congress of Paris, the "Semaine Odontologique Internationale", under the High Patronage of the President of the French Republic and under the Presidency of Honour of the Ministers of Public Health and of the Population, of National Education, of Industry and Commerce, of Labour and Social Security, will be held in Paris from Nov. 12–18, 1959 at the "Parc des Expositions", Porte de Versailles.

The vast exhibition of Dental Material and Products, unique on the French plan, which, at the last manifestation, covered an area of more than 7500 square metres, will include, as previously, an important foreign participation.

For all information, please apply to: M. Jacques Charon, Secrétaire Général de la "Semaine Odontologique Internationale", 31, rue Tronchet, Paris 8e.

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THE DENTAL PRACTITIONER and DENTAL RECORD

VOL. IX, NOS. 11, 12 JULY–AUGUST, 1959

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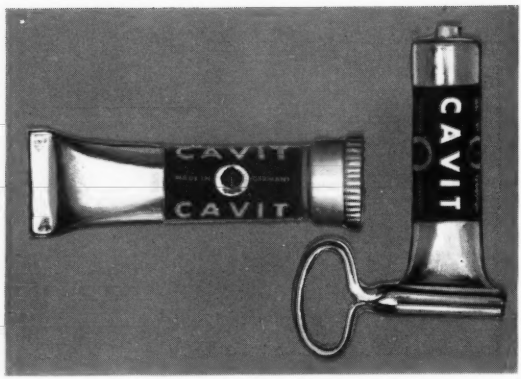

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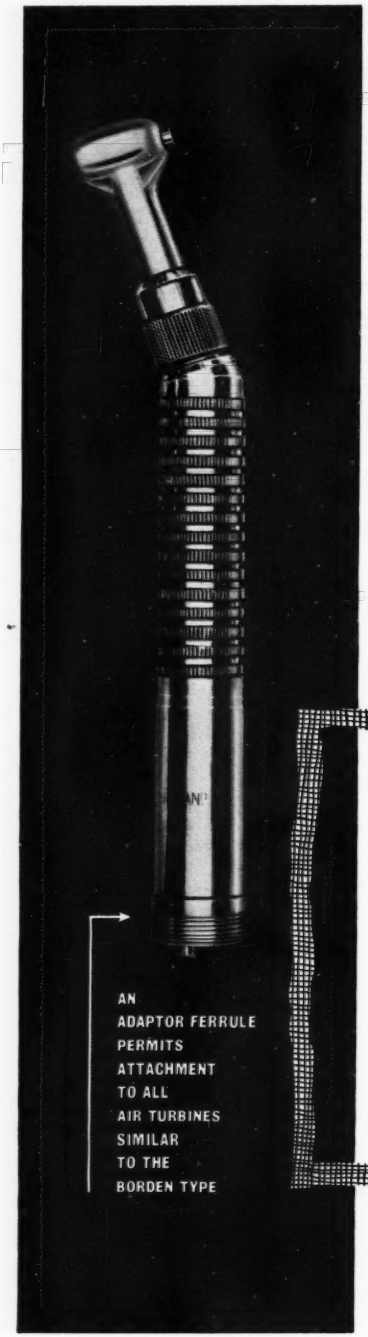

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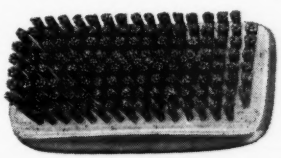

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